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THE
Journal of Obstetrics & Gynaecology
of the British Empire

VOL. 50, No. 6 NEW SERIES DECEMBER, 1943

A Review of the Problem of Purpura During Pregnancy

BY

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THE occurrence of purpura during pregnancy has been observed and studied for many years and yet the total number of recorded cases is very few. The first was described by Barnes[1] in 1867, since when a new case has been reported about once each year raising the total number to 75. Of these we have been able to trace descriptions of 68. The majority of these reports are presented with a paucity of clinical data and an absence of pathological facts which greatly detract from their value; blood counts are quoted in a minimum of cases, with the result that one is unable to appreciate either the type or the cause of the purpura described. At the same time, the early observers did not hesitate to quote mortality-rates for the disease and to formulate laws of its occurrence. We believe that many of these laws are only rough approximations to the truth, and we have taken the opportunity of describing a case of purpura complicating pregnancy which came into our care, and at the same time subjecting previous literature on the subject to a thorough analysis, in an endeavour to arrive at a truer conception of the features of this disease complex.

Purpura, even apart from pregnancy, is a condition which is still imperfectly understood though it can be classified into certain categories which most authorities find acceptable. Thus, as stated by Whitby and Britton,[2] there are three main types:

1. Purpuras showing quantitative deficiency in platelets (below 40,000 per c.mm.).

2. Purpuras with slight or no deficiency in platelets.

3. Purpuras due to a qualitative deficiency in platelets.

The first group comprises idiopathic essential thrombocytopenic purpura and malignant purpura due to bone marrow defect. In both conditions multiple haemorrhages occur into the skin or from the mucous membranes; bleeding time is prolonged, clotting time is normal and clot retraction is poor. In the essential form the reduced number of platelets may be caused either by an absence of maturation of megakaryocytes, which though present in normal

numbers themselves are inhibited by a substance derived possibly from the spleen or the platelets may be produced normally but destroyed as they circulate through the peripheral vessels and the spleen. Although the degree of purpura is proportional to the lack of platelets, the tone of the capillary walls also plays some part, as Macfarlane[3] has shown that the capillaries in such cases have a damaged endothelium, a defective contractility and malformed loops.

In the form known as malignant purpura, the reduction of platelets is due to a diminution in the number of magakaryocytes, often to an enhanced degree, which may occur alone or in association with a generalized bone marrow defect such as occurs with aplastic anaemia, medullary carcinomatous deposits, and with poisoning by benzol, novarsenobillon, etc.

The second group of purpuras comprises simple symptomatic purpura which is mainly due to a toxic degeneration of the vascular endothelium. Such drugs as belladonna, quinine and sulphapyridine may here be cited while a lack of vitamin C and vitamin P has the same effect. Anaphylactoid purpuras associated with urticaria, joint pains, oedema and visceral disturbances are included in this group. In these conditions the bleeding time, clotting time and clot retractility are normal.

Finally in the third group the platelets though normal in number are of deficient quality and disintegrate so easily as to cause purpuric symptoms. These conditions, typified by Glanzmann's haemorrhagic thrombasthenia and von Willebrand's thrombopathy, are essentially hereditary and encountered only infrequently.

When past observers have collected a series of cases of purpura of pregnancy, they have made no attempt at classification. Thus they have grouped together purpuras of all three types and have drawn conclusions from them as a whole. Rushmore[4] in 1925 stated that the mortality-rate of purpura of pregnancy was 59 per cent for the mother (out of a total of 44 cases) and 64 per cent for the child (42 cases); he also concluded that it was more frequent in multiparae (28 out of 35 cases), and occurred chiefly during the later months of pregnancy (28 were in the last 3 months out of 38 cases). These conclusions we venture to suggest are but statistical averages of the true facts. The incidence, prognosis and mortality-rate of purpura will differ, in the same manner as statistics concerning any particular symptom will differ, according to the type of purpura concerned. In an endeavour to paint a more correct picture we have classified the 68 traced cases into the above three categories of Whitby and Britton, and from a study of each group separately we can hope to obtain information of a more helpful and accurate character. In our attempt to do this we have met with great difficulty, as the platelet counts have been recorded only 12 times, and the bleeding and clotting times still less frequently. However, in spite of this, the clinical histories are in some cases so detailed as to leave no reasonable doubt as to the cause of the purpura. Table I contains 22 cases of purpura of pregnancy in each of which the cause of the purpura has been ascertained with a fair degree of accuracy; the remaining 46 we have had to omit as being of too doubtful aetiology.

Purpura is well known to be a rare association of pregnancy, but a study of Table I reveals the extreme rarity of true essential thrombocytopenic purpura even among the recorded cases. Only 4 of the 68 cases can be accepted as examples of this condition. Cases exhibiting qualitative platelet defect are also rare as would be expected from the infrequency with which these conditions are encountered. It is interesting to note incidentally that cases have not been reported from the Baltic area, for it is there that

TABLE I.

GROUP I.	Case	Year	Parity of mother	Duration of pregnancy	No. of platelets	Fate of mother	Fate of child
(a) Essential thrombocytopenic purpura	1. Liebling[5]	1926	1	5 months	40—300,000	Full recovery in 3 months	Full recovery in 3 months.
	2. Siegler[6]	1934	5	Postmature	80,000 BT—1 minute CT—1½ minutes CRT—2½ hours	Recovered	Died at 4 days
	3. de Saussure and Townsend[7]	1935	9	Term	33,000 BT>30 minutes CT—5½ minutes	Full recovery	Stillborn
	4. Bernstein, Newman and Hitzig[8]	1939	1	5 months	10,000 BT>10 minutes CT—13 minutes	Full recovery	Normal, but died from atelectasis
(b) Bone marrow defect	5. Posner[9] (due to quinine)	1937	2	Postmature	17,000	Full recovery	Purpura at birth Platelets, 10,000 Recovered later
GROUP II.							
(a) Anaphylactoid.	6. Barnes[1] (associated with rheumatism)	1867	2	6 months	—	Died	Died
	7. Rushmore[4] (associated with urticaria)	1925	1	6 months	—	Full recovery	Stillborn No purpura
	8. Long and Orr[10] (associated with rheumatism)	1940	2	Term	43,500 (very anaemic)	Recovered	Normal
(b) Symptomatic	9. Vignes and Stiassnie[11] (associated with syphilis)	1921	3	Term	—	Recovered	Normal
	10. Peshkin and Miller[12] (due to quinine)	1934	3	2 months	130,000 BT—3 minutes CT—4 minutes	Recovered	Therapeutic abortion
	11. McCoogan[13] (anti-syphilitic treatment)	1935	2	7 months	158,000	Recovered	Stillborn (due to accidental haemorrhage)

TABLE I—(*Continued*)

GROUP II—Continued	Case	Year	Parity of mother	Duration of pregnancy	No. of platelets	Fate of mother	Fate of child
	12. Stone and Bunim[14] (due to quinine)	1936	1	Term	Normal BT—2½ minutes CT—3 minutes	Recovered (followed by acute yellow atrophy)	—
	13 Israel and Winsom[15] (due to hyper-thyroidism)	1941	1	6 months	140,000 BT—normal CT—normal	PPH recovered	Stillborn triplets
(c) Due to fevers	14. Wiener[16] (fever unknown, probably smallpox)	1887	1	7 months	—	Died	Died
	15. Hanot and Luzet[17] (Streptococcal septicaemia and meningitis)	1891	1	Term	—	Died	Died
	16. Leclerc[18] (fever unknown but polymorphs 86 per cent)	1908	5	6 months	—	Died	Died
(d) Due to scurvy	17. Ferroni[19]	—	—	—	—	Recovered	Normal
	18. Zangemeister[20]	1898	1	6 months	—	Recovered	Died
	19. Mosher[21]	1923	6	7 months	—	Recovered	Died
GROUP III.	20. Waltner[22]	1924	1	7 months	—	Recovered	Purpura at birth
(a) Qualitative platelet defect	21. Sanford, Leslie and Crane[23]	1936	1	Term	10,000	Healthy but platelets low	Purpura at birth. Platelets 20,000
	22. Davidson[24]	1937	1	Term (8½ years after splenectomy)	6—47,000 BT—58 minutes CT—1½ minutes	Recovered. Platelets 21,000 6 months later	Platelets 28,000 Normal at 2½ years

most observations of these diseases have been made.

The most common types of purpuric changes that occur in pregnancy are the symptomatic purpuras, appearing in association with rheumatism, drug intoxications, fevers and scurvy. The last disease must have been a common cause of purpura of pregnancy in past decades, for Mosher[21] writing in 1923 describes what Rushmore calls "the best composite picture of the clinical course of pregnancy complicated by purpura" as follows:

"Her gestation shows no untoward symptoms until some time in the 6th or 7th month she experiences a vague discomfort, loses appetite and complains of headache; she has palpitation and gastro-intestinal trouble which becomes more marked. Some time later there appear haemorrhagic spots on the skin, then petechiae, which are at first discrete, but later become confluent. These spots usually appear in successive crops. The woman finds that her gums are tumefied and painful and begin to bleed; epistaxis becomes troublesome; she usually has a persistent diarrhoea; she consults a doctor who finds that the foetus is living but the mother has purpura with symptoms of grave character."

It is evident that this is a description of scurvy in a pregnant woman, and cannot be accepted therefore as an example merely of purpura. In view of recent knowledge gained as to the prevalence of sub-clinical scurvy at the present day, it may be concluded that many of the past cases were due to this cause. Thus it is probable that one of Ferroni's[19] cases and those of Zangemeister[20] and Mosher[21] were of this nature; there is little doubt that a number of the 46 unclassified cases also belong here, as it can be fully appreciated that the extra strain of pregnancy thrown on a mother near the borderline of vitamin C insufficiency would easily precipitate her into a scorbutic state, and produce what Rushmore quotes as a "typical" example.

In a similar manner the case described by Hanot and Luzet[17] cannot be considered to be primarily a case of purpura of pregnancy; it is essentially a case of streptococcal meningitis and septicaemia with purpura, in which the pregnancy is purely incidental. In the cases described by Vignes and Stiassnie[11] and McCoogan,[13] drugs used for anti-syphilitic treatment appear to have been the causal factor, while in those reported by Posner,[9] Peshkin and Miller,[12] and Stone and Bunim,[14] quinine has apparently exerted a noxious influence on the bone marrow, even producing in Posner's case an actual thrombocytopenia of 17,000. Many of the unknown cases probably resulted from the use of these drugs, owing to the past higher incidence of syphilis, and the extensive use made of quinine as an abortifacient and in medical inductions of labour.

We have recently had under our care a case of purpura of pregnancy which we have been able to study in some detail, and which we believe to be an example of true thrombocytopenic purpura. As this is only the fifth case which we can discover since 1867, we feel it will be of advantage to describe the clinical picture and the pathological features which it presents.

Case Report.

Mrs. J. W. was a primigravida, aged 21, who first attended the antenatal clinic on June 6th, 1942, when she was 11 weeks pregnant. Her previous history was good, and did not contain anything of clinical note, and abnormal clinical findings were not present on examination. The family history was equally uneventful, except for the fact that her father had contracted malaria during the last war. The husband's family and personal medical histories were similarly devoid of clinical interest.

J.W. next attended the clinic on June 30th, 1942, when she was 14 weeks pregnant, and complained

of epistaxis which had commenced 4 days previously and had lasted for 2 days. Her general health was good and there had not been any morning vomiting. Examination revealed extensive and widespread ecchymoses and crops of petechiae present on the chest, abdomen, arms, thighs and ankles; all of these had spontaneously appeared about the same time as the epistaxis. Intensive questioning did not reveal any history of a previous similar attack, and detailed enquiry as to her diet did not show a deficiency of any of the food values. There was no evidence of pregnancy toxaemia. The patient was admitted for investigation and treatment.

In hospital close clinical examination failed to reveal any abnormal physical signs apart from those already mentioned. The circulatory, respiratory and nervous systems were normal, the mouth and gums did not show any pathological changes, the spleen was not palpable, and there were no enlarged lymphatic glands to be felt. The patient had not been taking any drugs. Her blood-pressure was 124/75 mms. Hg. and her blood count on admission was as follows:

Red-blood cells, 5,060,000 per c.mm.; white-blood cells, 8,000 per c.mm.; differential count normal; haemoglobin, 82 per cent; colour index, 0.82; platelets, 15,000 per c.mm.; no abnormal forms seen.

Bleeding time (BT), 27½ minutes; clotting time (CT), 4 minutes; Clot retractility, delayed; Tourniquet test, strongly positive.

A provisional diagnosis of thrombocytopenic purpura complicating pregnancy was made, but active treatment was not instituted as further bleeding did not occur. The following investigations were carried out with the results shown:—

Wassermann reaction, negative; plasma fibrinogen, 310 mgm. per cent; blood ascorbic acid, 0.47 mgm. per cent; blood prothrombin time, 3 minutes 45 seconds (Howell); red-cell fragility, within normal limits; blood calcium, 12.8 mgm. per cent.

Sternal puncture showed a normal picture (see below). Microscopy of the urine revealed some red-blood cells, and there was a trace of occult blood in the faeces. Harris and Ray's ascorbic acid saturation test showed no deviation from normal, the results being similar to those obtained in three apparently healthy controls. Unfortunately practical difficulties prevented us from carrying out estimations of pregnandiol excretion.

As there were no further haemorrhages and the original ecchymoses had regressed the patient was discharged, advised to take supplementary vitamin therapy, and kept under supervision at the antenatal clinic. Progressive platelet counts were carried out and the results are shown in Table II.

TABLE II.

Date	Period of pregnancy, in weeks	Platelet count, per c.mm.	Tourniquet test
July 2nd, 1942	14	15,000	Positive
July 3rd, 1942	14	Practically absent	Strongly positive
July 10th, 1942	15	21,000	Positive
July 23rd, 1942	17	62,000	Positive
August 6th, 1942	19	102,000	—
August 12th, 1942	20	145,000	Positive
September 8th, 1942	24	138,000	—
October 20th, 1942	30	160,000	—
November 10th, 1942	33	127,000	Positive
November 28th, 1942	35	163,000	—

Apart from the regular appearance of fresh petechiae and ecchymoses the patient's health remained excellent during the later months of her pregnancy and no further external bleeding occurred.

On January 2nd, 1943, the patient was delivered of a healthy female child, weighing 7 pounds 14

ounces, following a labour lasting for only 6 hours with a 1st vertex presentation. Despite a perineal laceration of the second degree the mother's postpartum loss was slight. The infant bore no evident stigmata of purpura and the placenta appeared normal. At the time of delivery the mother's platelets were 76,000 and those of the placental blood were stated to be in excess of 121,000, an accident preventing the true figure being ascertained. Afterwards mother and child progressed satisfactorily and the puerperium was normal. At the end of the first week the mother's platelets had risen to 105,000, bleeding time was 4 minutes, clotting time 4 minutes, and the tourniquet test was weakly positive. The infant's platelets were then 332,000. She was discharged from hospital on the 12th day after delivery, when the perineum was healed, the lochia were scanty and colourless and the uterus was anteverted and normally involuted. Petechiae were not present and breast feeding was well established. The umbilical cord had separated on the 6th day, the umbilicus was healed, and the weight of the child on discharge was 7 pounds 10 ounces.

Seven weeks after delivery the mother was seen at the postnatal clinic, when, although she was obstetrically normal and still had amenorrhoea, a few petechiae were visible on the abdomen and thighs, and she stated that she had had slight epistaxis. A blood count taken at this time revealed that her platelets were 162,000; later, 3 months after delivery, when her symptomatic recovery was quite complete, they were 174,000. The baby was then thriving normally, and had a platelet count of 345,000.

Conclusions.

There can be no doubt that this case was one of true thrombocytopenic purpura complicating pregnancy, for in addition to the diminished number of platelets the bleeding time was prolonged, the clotting time was normal and clot retraction was poor. The clinical course of the disease and the normal appearances upon sternal puncture exclude the possibility of malignant purpura, while the absence of peripheral symptoms eliminate a purpura of the anaphylactoid type. No noxious drugs had been taken, no fever had occurred, there was no syphilis, the blood ascorbic acid test and the vitamin C saturation tests rule out the possibility of scurvy. The condition was not inherited and no question of Glanzmann's thrombasthenia or allied complaint can be raised. In addition the blood chemistry was normal, as the blood calcium, fibrinogen, prothrombin and cell fragility all show.

The salient features of the 4 recorded cases of true essential thrombocytopenic purpura, those of Liebling,[5] Siegler,[6] de Saussure and Townsend,[7] and Bernstein, Newman and Hitzig[8] are included in Table III for comparison with the present case.

From the facts portrayed in Table III and those presented in the case report it is possible to form the following conclusions:

Family history. Antecedent family history of purpura was not apparent in any case; in one instance only was it stated that the patient's mother bruised easily.

Age and Parity. The condition can occur at any age within the child-bearing period, and in association with any degree of parity.

Health prior to pregnancy. In only one case (the one in which the mother's bruising occurred) was any haemorrhagic tendency noted.

Health in previous pregnancies. In both cases noted this was normal.

Health during reported pregnancy. In one case petechiae were first observed in the early weeks and haemorrhages occurred later in the pregnancy; in our case at 14 weeks; in 2 cases at 5 months; in only 1 case did the bleeding start at term. The number of platelets varied considerably; in Liebling's case and in ours they fell in mid-pregnancy and rose again during the third trimester, in spite of the absence of very active therapeutic measures. In Bernstein, Newman and Hitzig's case they rose similarly but only in the customary manner after splenectomy. Contrary to their con-

TABLE III.

CASE	LIEBLING	SIEGLER	DE SAUSSURE and TOWNSEND	BERNSTEIN, NEWMAN, and HITZIG
Family history	Negative for haemorrhagic tendency	Mother bruised easily	Not stated	Not stated
Age	22	29	35	23
Health prior to pregnancy	Not stated	Menorrhagia. Subject to black and blue spots for 7 to 8 years	Not stated	Bilateral otitis media and sinusitis Periods normal
Parity	1	5	9	1
Health in previous pregnancies	Not stated	Postpartum haemorrhage after 4th labour otherwise normal	Normal	Not stated
Health during pregnancy	Petechiae appeared at 5 months, with bleeding from gums and epistaxis. Platelets 40,000 at 6 months; 300,000 at 7 months	Spots noticed by patient at 5th month. Not seen by doctor until at delivery. Platelets 80,000 BT—1 minute CT—1½ minutes CRT—2½ hours	Bleeding from mouth and vagina on day before delivery. Platelets 33,000. BT>30 minutes CT—5½ minutes.	Petechiae in early weeks. Vaginal haemorrhage and epistaxis at 5th month Platelets 10,000 BT>10 minutes CT—13 minutes Splenectomy at 24 weeks. Platelets then rose to 220,000
Labour	Normal. Amount of bleeding not stated	Postmature. No tear. Loss of 1½ litres of blood.	Normal. Moderate bleeding.	Two months premature. Normal, 30 c.c. blood lost
Puerperium	Platelets 20,000 on 4th day BT—4½ minutes CT—5 minutes	Petechiae faded by 5th day	Platelets 52,000 after delivery	Normal. Daily blood-counts normal
Child	Platelets 40,000. Petechiae present at birth. These faded no new ones appeared	Died at 4th day. No postmortem or blood count recorded. Presence of purpura not stated.	Stillborn. Presence of purpura not stated	Normal but premature Platelets 170,000 Died at 8 days from atelectasis.
Follow-up	Petechiae present on mother up to 2 months postpartum Fully recovered at 3 months	Platelets 80,000 at 6 weeks. 120,000 at 22 weeks.	Platelets 130,000 in 4 weeks; 237,000 in 26 weeks	Platelets 180,000 at 43 days Thereafter, full recovery
Treatment	Ca lactate and ferrous carbonate	30 c.c. husband's blood. Liver extract. Sunlight. Haematinic diet.	Horse serum. X-ray treatment. Antivenin. Intravenous glucose. Iron, copper and arsenic.	Blood transfusions. Iron and vitamins A, B, D. Moccasin snake venom. Splenectomy at 24 weeks

clusion, it is evident that improvement in the blood picture can occur apart from this operation. In the case described by Siegler, although the patient noticed spots at the 5th month, when presumably the platelets must have been few, a count was not made until delivery, when in the absence of any previous treatment they were 80,000. The thrombocytopenia thus usually appears early in the pregnancy.

Labour. The amount of blood lost at the confinement varies from case to case; apparently haemorrhage may occur, but in no case is it very alarming—in our patient, despite a perineal laceration, it was slight. The haemostatic effect of efficient uterine retraction is evidently still the factor of paramount importance in these thrombocytopenic cases.

The puerperium. After delivery in all cases the petechiae faded and the platelet count rose, though in 2 cases, Liebling's and ours, fresh petechiae did appear during the first 2 months; however, within periods varying from 3 to 6 months all observers record the mother's full recovery.

The child. No rule can be laid down about the fate of the child. Of 5 cases 3 infants died, but apparently not from purpura. Siegler's case may be an exception, as he states that the probable cause of death was purpura haemorrhagica, but he does not record that petechiae were present, no blood count was made and no postmortem examination was performed. The case we have reported shows that it is quite possible for the pregnancy to terminate in the birth of a healthy child with a normal blood count.

The reported cases in which the child develops purpura (Rushmore quotes 7 out of 47 cases) are probably either examples of symptomatic purpura in which the child is subjected to the same influence as the mother, such as quinine in Posner's case, or else they are examples of familial platelet defect.

Treatment. All 5 cases had completely different treatment and all recovered. Probably, therefore, the treatment has little influence on the complaint. Bernstein *et al.* form the conclusion that splenectomy is necessary for the birth of a healthy child; our case shows that this is not so. They also suggest that the absence of postpartum haemorrhage is due to the splenectomy, a fact which our case again disproves. It thus appears that the indications for this operation are the same as obtain in purpura apart from pregnancy.

Sternal puncture. Sternal punctures are recorded in only 2 cases with results as follows:

BERNSTEIN, NEWMAN and HITZIG

Cell count	250,000
Megakaryocytes	13.2 per cent
Myeloblasts	1.4 ,,
Myelocytes	29.4 ,,
Polymorphonuclear leucocytes	48.8 ,,
Lymphocytes	1.0 ,,
Other cells	6.2 ,,

PRESENT CASE

Cell count. Normal cellularity not very active

Megakaryocytes 3/500 nucleated cells

Myeloblasts	1.0 per cent
Myelocytes	7.4 ,,
Polymorphonuclear leucocytes	50.4 ,,
Lymphocytes	20.4 ,,
Eosinophils	1.6 ,,
Metamyelocytes	5.2 ,,
Plasma cells	0.2 ,,
Monocytes	0.6 ,,
Normoblasts	11.8 ,,
Pro-erythroblasts	1.2 ,,
Haemocytoblasts	0.2 ,,

Apart from the excess of megakaryocytes in the former case both counts are stated to be within normal limits.

DISCUSSION.

The relation between purpura and pregnancy still remains obscure. We do not know whether the patients exist all their lives with a low platelet count which the strain of pregnancy reduces to below the purpuric level of about 40,000, or whether they are completely normal individuals who are subjected to a pathological process during their pregnancy which produces purpura. As the strain of previous pregnancies in the multiparae does not appear to have precipitated purpura, it seems likely

that the latter contention is the correct one. In either case we have no idea what this "strain" or pathological process that produces the thrombocytopenia actually is. It is true that earlier workers have reported examples of recurrent purpura of pregnancy but on close examination these have proved to be cases of symptomatic purpura, such as the case of Vignes and Stiassnie[11] in which purpura occurred during 3 successive pregnancies in a syphilitic woman. In hereditary purpura also the purpura is recurrent; thus Sanford *et al.*[23] and Davidson,[24] in presenting cases of qualitative platelet defect, conclude that the platelets possess an increased disintegrative ability and so produce purpura when the patient is subjected to increased strains of life such as pregnancy. In these cases, however, the condition is familial in a manner that true purpura is not.

We must also consider the possibility that the purpura and the pregnancy are unrelated causally, and that the patient who suffers from purpura haemorrhagica and is subject to periodic relapses chances to have one which coincides with pregnancy. The extreme rarity of the association somewhat suggests this view, but as each of the 5 cases did not give any history of a previous relapse, and each made a complete symptomatic recovery within 3 to 6 months after delivery, it is impossible not to conclude that the two conditions are correlated in some manner. Observation of the subsequent progress of these cases might however reveal that the recovery is transient and that further relapses are liable to occur; in this event the association with pregnancy must be considered fortuitous. It is true no such case has yet been reported to the best of our belief, but until these accepted cases have been watched for many years and their continued recovery confirmed, we cannot deny that such an occurrence is possible.

Some authors speak of purpura being due to a toxaemia of pregnancy, Kosmak[25] and Seitz[26] stating that it occurs in association with hyperemesis gravidarum and eclampsia. This association must be most rare in view of the great frequency with which these two toxaemias are encountered, and moreover in those cases which have been reported the purpura is of the non-thrombocytopenic variety. Purpura occurring in toxaemia of pregnancy is of the symptomatic type, and whether the two are causally related or purely coincidental is problematical, and does not concern our consideration of true purpura, in cases of which, as we have shown, evidence of these toxaemias is completely lacking. Liebling suggests that his case may be due to toxaemia but offers no support for his statement. If we do accept a toxaemia theory we must hypothecate an independent form of metabolic toxaemia, such as what Siegler terms a "latent toxaemia," affecting only the platelets. We do not ourselves accord much support for such an assumption, because not only is its occurrence extremely rare, but the purpura begins very early in pregnancy and has a distinct tendency to improve in the absence of treatment as the pregnancy progresses, which is unusual for a toxaemia.

An interesting speculation has been opened up by the work of Pohle[27] who has shown conclusively that the platelet count varies with menstruation, being lowest before the onset of the bleeding. After careful analysis he suggests that the cause of this may be hormonal in nature. If this be so it is evident that the increased hormonal fluctuations of pregnancy may in certain circumstances reduce the platelet count still further and give rise to the onset of purpura. Pfeiffer and Hoff[28] have demonstrated that a thrombocytopenia may result from the action of progesterone on the spleen, and should this hormone be produced in quantities greater than the normal for preg-

nancy, we suggest it may cause a reduction to purpuric levels. It is for this reason we regret our inability to carry out pregnandiol excretion estimations in our case. The early occurrence of thrombocytopenia and its tendency to disappear later in pregnancy, suggest that it is those hormones which reach a high concentration in the early months, such as the APL principle and the pituitary gonadotrophins, that may be responsible. In view of the normal megakaryocyte count obtained on sternal puncture we must assume that the platelet defect whatever the cause must result from either a faulty maturation of megakaryocytes which are themselves present in normal numbers, or else from their destruction as they circulate through the peripheral blood stream.

In this respect we must mention the work of Sakai[29] who considers that reduction in the platelet count before menstruation results from increased activity of the reticuloendothelial system. Hyperactivity of this system during pregnancy might thus contribute to the occurrence of purpura.

Although these cases are too few in number to form a basis for definite conclusions, they do cause our views to diverge from those formulated by Rushmore in his study of purpura of pregnancy due to all causes, which have been generally accepted. These 5 cases of true purpura do not support his contention that purpura is more frequent in multiparae as 3 of them occurred in primigravidae. Again they do not conform with his opinion that purpura usually commences during the last 3 months of pregnancy, as we have demonstrated. He quotes a maternal mortality-rate of 59 per cent, de Saussure and Townsend quote one of 55·7 per cent, while in these 5 cases all the mothers recovered completely, so that true purpura of pregnancy does not appear to be a fatal disease. Certainly we do not agree with Hirst[30] who states that the disease is generally fatal and always interrupts pregnancy, the foetus dying *in utero*.

We suggest, therefore, that the conclusions reached in the past do not apply to true essential thrombocytopenic purpura of pregnancy which differs diametrically from them in practically all respects; the correct picture of this complaint has in the past been disguised by the superadded features of other diseases which have been present merely as incidental causes of symptomatic purpura.

Summary.

1. An analysis of 68 recorded cases of purpura of pregnancy is made and in 22 cases the cause of the purpura is ascertained.

2. Only 4 cases of true essential thrombocytopenic purpura complicating pregnancy can be discovered.

3. A fifth case of true purpura is presented.

4. The features of these 5 cases are reviewed, and conclusions are reached regarding the incidence, clinical course, treatment and mortality of the condition.

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Blood Examinations in Pregnancy

BY

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IN this paper the results of blood studies on a group of 105 women attending one of the Fulham Antenatal Clinics and the Fulham Nutrition Clinic between the end of 1936 and the middle of 1939 are reported. They were unselected. All belonged to the working and lower middle classes. Eleven cases could not be included in the general summary—6 suffered from concomitant diseases; 3 came from other clinics; 2 left the district before labour—leaving 94 cases. Of these 55 were primiparae and 39 multiparae. They were between 19 and 37 years of age. A detailed report will be published by the Metropolitan Borough of Fulham.

METHODS.

Haemoglobin examinations were made with a Haldane haemoglobinometer standardized at an oxygen capacity of 18.5 per cent. Red and white cells were counted in a Thoma-Zeiss camera. The films were fixed and stained with Jenner-Giemsa solution. Red cells were dyed when wet by the azure-cresyl method.[1] Only the size of fresh wet unfixed red cells not more than slightly stained yellow was estimated because neither a Price-Jones apparatus nor a Wintrobe's haemotocrit was available, and also because, in accordance with Boycott's observations,[2] different results were found in different parts of the stained films.

The aim of the investigation was to establish the normal physiological standards for the whole blood picture: haemoglobin, red cells, colour index and white cells, and size of the red cells; to establish their mean values for all periods of pregnancy, puerperium, and of the nursing and post-nursing period; and to investigate the frequency and type of the anaemias in pregnancy with regard to their aetiological agent and their response to treatment.

PHYSIOLOGICAL DATA.

Any attempt to establish normal physiological standards can only be based on non-treated cases. *The series of* 94 *cases* therefore was divided into three groups as follows:

GROUP I.

Cases non-treated with iron, protein, or liver during pregnancy or puerperium — 43

Cases showing haemoglobin under 70 per cent more than once during pregnancy were not included in this group as it is generally agreed that they cannot be regarded as normal.[3, 5, 12]

GROUP II.

Cases treated in the antenatal clinic, before first attendance at the nutrition clinic, and throughout pregnancy with routine administration of iron ... — 7

4 Blaud's pills, gr. xxx, were given daily because they did not feel well.

GROUP III.

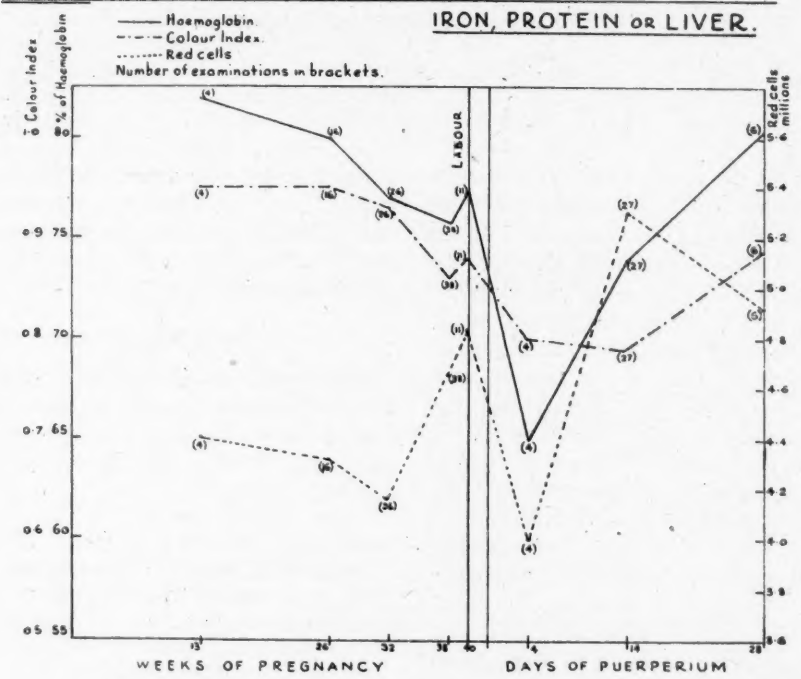
Cases treated with iron, protein, or liver in different periods of, or throughout pregnancy ... ... — 44

The mean values of cases in Group III are not given because their blood picture showed different specific variations either at their first attendance or later in pregnancy, and received specific treatment accordingly.

94

GRAPH I

GROUP I: 43 CASES NON TREATED DURING PREGNANCY OR PUERPERIUM WITH IRON, PROTEIN OR LIVER.



Whole Series.

Mean figures for the whole series of 94 cases, both treated and not treated, are given for comparison with the findings of authors who do not give separate accounts of treated and non-treated women during pregnancy.

Mean values are given in Tables I, II, and III, and in Graphs I, II and III.

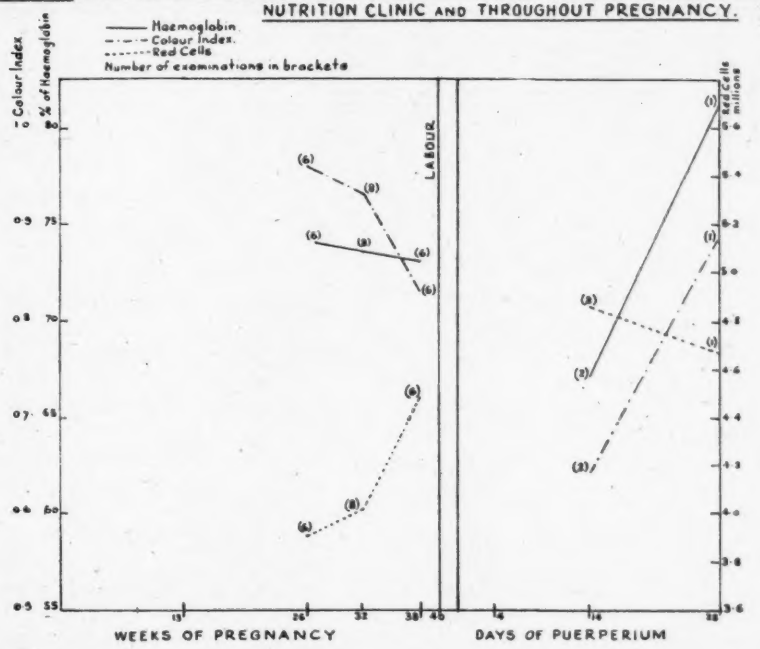
Values in Pregnancy.

The results in pregnancy in general show, in agreement with Fullerton,[4] that the haemoglobin drops with progress to term, and only rises in the 2 weeks before labour. The red cell count drops similarly in the first 6 months of pregnancy, but rises markedly and continuously in the last 3 month period taken as whole until labour. The course of the haemoglobin and the red cell count are therefore not parallel in the last 3 month period of pregnancy. The colour index is lower in the last 3 months of pregnancy than in the first 6 months, and only recovers with the rise of the haemoglobin in the last 2 weeks before labour. Owing to the sudden sharp rise of the red cell count in the last 3 month period and the slow fall of the haemoglobin, the colour index drops substantially. The white cells are increased with progress to term up to 20,000–25,000 in conformity with the results found by other authors,[5] and are therefore not given separately.

In order to show more clearly the differences in the non-parallel course of the haemoglobin and the red cells in the whole last 3 month period before labour, separate figures are given for the following periods: (1) 27th to 32nd weeks; (2) 33rd to 38th

GRAPH II.

GROUP II: 7 CASES TREATED WITH ROUTINE ADMINISTRATION OF IRON BEFORE ATTENDANCE AT NUTRITION CLINIC AND THROUGHOUT PREGNANCY.



weeks; (3) 39th to 40th weeks. Some significant variations of the red cell count in the last 3 months of pregnancy will be discussed later, but the actual rise of the red cells starts in most cases in the 33rd week and goes on until labour.

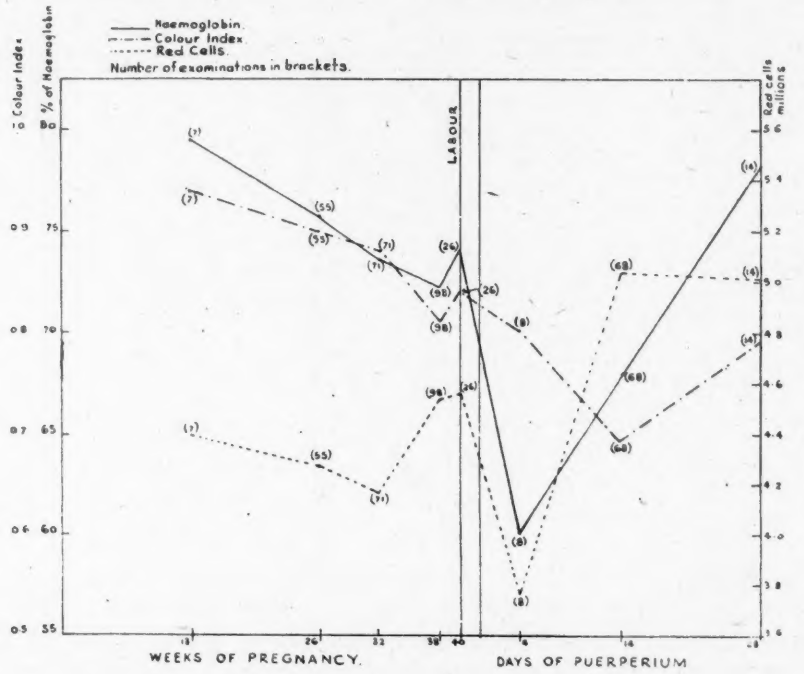
VALUES IN DIFFERENT PERIODS OF LAST 3 MONTHS

MEAN VALUES.	For whole period of pregnancy	For last 3 months	Week 27–32	Week 33–38	Week 39–40
GROUP I.					
Red cell count	4.43 (95)*	4.41 (75)	4.18 (26)	4.45 (38)	4.84 (11)
Haemoglobin	78.5 per cent	76.5 per cent	77.1 per cent	75.9 per cent	77.4 per cent
Colour index	0.91	0.88	0.93	0.86	0.88
GROUP II.					
Red cell count	4.14 (20)	4.24 (14)	4.03 (8)	4.49 (6)	—
Haemoglobin	73.6 per cent	73.0 per cent	73.8 per cent	73.0 per cent	—
Colour index	0.91	0.85	0.92	0.83	—
WHOLE SERIES.					
Red cell count	4.39 (257)	4.36 (195)	4.17 (71)	4.54 (98)	4.55 (26)
Haemoglobin	75.2 per cent	73.2 per cent	73.5 per cent	72.3 per cent	74.0 per cent
Colour index	0.87	0.83	0.88	0.81	0.84

* Figures in parenthesis in all tables refer to number of examinations.

GRAPH III.

WHOLE SERIES: 94 CASES TREATED OR NON TREATED IN PREGNANCY WITH IRON, PROTEIN OR LIVER.



VALUES IN PARTURITION.

During parturition the values of the haemoglobin and the red cell count in Group I (4 cases), and in the Whole Series (8 cases), drop substantially in the first 4 days after labour. No cases from Group II happened to be seen during the first 4 days after labour (see Tables I, II and III and Graphs I, II and III). It is well known[4 18] that the Hb and R.C.C. fall directly after labour. In this period only a few cases could be examined, which included patients with post-labour haemorrhage. After the first period of 1 to 4 days after labour the red cell count rises again between the 5th and 14th days inclusive after labour even above the level for the last 3 months of pregnancy; the haemoglobin level also rises, but more slowly. This *divergent movement* leads again to a lowering of the colour index which falls below the antepartum value, and rises again with the return to normal, i.e. the rise of the haemoglobin and the decrease of the red cells between the 15th and 28th days after labour. In these two periods of the puerperium (5th to 14th days and 15th to 28th days) the rate at which the haemoglobin and the red cell count return to normal and the colour index to unity can be considered as indices of the rate of recovery. Both haemoglobin and red cell count are tending back to normal mean values in the nursing and post-nursing periods, but in some cases the mean values in the lactation period proved to be equal to or even lower than those found in pregnancy, showing

GROUP I—TABLE I.

ALL CASES NON-TREATED DURING PREGNANCY OR PUERPERIUM WITH IRON OR PROTEIN OR LIVER.

Pregnancy						Puerperium: Days postpartum		
Week	12–13	14–26	27–32	33–38	39–40	1–4	5–14	15–28
Red cell count	4.41	4.32	4.18	4.45	4.84	4.01	5.30	4.93
Haemoglobin (per cent)	82.2	80.0	77.1	75.9	77.4	65.0	74.0	80.5
Colour index	0.95	0.95	0.93	0.86	0.88	0.80	0.69	0.89
No. of examinations	(4)	(16)	(26)	(38)	(11)	(4)	(27)	(6)

MEAN VALUE OF ALL 43 CASES AND ALL PERIODS OF PREGNANCY:

Red cell count 4.43 (95)
Haemoglobin ... ... 78.5 per cent (95)
Colour index 0.91 (95)
Total cases in pregnancy: 43. Total of examinations: 95.

MEAN VALUE OF DIFFERENT PERIODS OF PREGNANCY.

	Red cell count	Haemoglobin	Colour index
Mean value of first 6 months	4.34 (20)	80.5 per cent	0.94
Mean value of last 3 months	4.41 (75)	76.5 ,,	0.88
Mean value, 39th–40th weeks	4.84 (11)	77.4 ,,	0.88
PUERPERIUM: Days postpartum:			
1–4	4.01 (4)	65.0 ,,	0.80
5–14	5.20 (27)	74.0 ,,	0.69
15–28	4.93 (6)	80.5 ,,	0.89
Total of cases in puerperium 30	Total of examinations 37		

that recovery after delivery is not necessarily complete. This is in agreement with Mackay.[6]

SIZE OF RED CELLS.

The red cells in Group I were found to be constantly of average and equal size in the first 6 months of pregnancy; in the last 3 month period, with the increase in the number of the red cell count, new large cells and even megalocytes appeared, some of which were poorly haemoglobinized and showed central pallor. Anisocytosis was frequent, but poikilocytosis rare if at all. During the puerperium the cells became smaller again and more equal. The gradual return to the normal size of cells was completed in 8 to 9 months after labour, provided that pathological disturbances did not occur in the nursing or post-nursing periods. Reticulocytes were never increased in the non-treated cases. The differences found in the cells in anaemic cases will be discussed later.

DIFFERENCES IN THE MEAN VALUES OF DIFFERENT GROUPS DURING PREGNANCY AND PUERPERIUM.

The general trends of haemoglobin, red cell count, and colour index are the same in all groups. The mean fall in the haemoglobin level of Group I (non-treated) is 6.3 per cent between the 3rd and 8th months, with a recovery before term of 1.5 per cent. The corresponding changes for the Whole Series are a fall of 7.5 per cent and a rise of 1.7 per cent. The cases treated with routine administration of iron in Group II showed a drop of 1 per cent. They were only seen between the 4th and the 8th months of pregnancy, not between the 39th and 40th weeks before labour, and therefore for these weeks figures cannot be given. The routine

GROUP II—TABLE II.

CASES TREATED WITH ROUTINE ADMINISTRATION OF IRON BEFORE FIRST ATTENDANCE AND THROUGHOUT PREGNANCY.

Pregnancy						Puerperium: Days postpartum		
Week	12–13	14–26	27–32	33–38	39–40	1–4	5–14	15–28
Red cell count	0	3.90	4.03	4.49	0	0	4.86	4.67
Haemoglobin (per cent)	0	74.0	73.8	73.0	0	0	67.0	81.0
Colour index	0	0.96	0.92	0.83	0	0	0.64	0.88
No. of examinations		(6)	(8)	(6)	(0)	(0)	(2)	(1)

MEAN VALUE OF ALL 7 CASES AND ALL PERIODS OF PREGNANCY.

Red cell count	4.14 (20)
Haemoglobin	73.6 per cent (20)
Colour index	0.91 (20)

Total of cases, 7. Total of examinations, 20.

MEAN VALUE OF DIFFERENT PERIODS OF PREGNANCY.

		Red cell count	Haemoglobin	Colour index
Mean value of 2nd 3 months	(6)	3.90	74 per cent	0.96
Only 27th–38th weeks of last 3 months	(14)	4.26	73 ,,	0.86
39th–40th weeks	(0)	—	—	—
PUERPERIUM: Days postpartum:				
1–4	(0)	—	—	—
5–14	(2)	4.86	67 per cent	0.64
15–28	(1)	4.67	81 ,,	0.86

Total of cases in puerperium, 3. Total of examinations, 3.

administration of 4 Blaud's pills daily to these patients prevented a further drop of the haemoglobin with progressive term but the red cell count remained in the first 6 months below the values of Group I. In the values of the red cell count in Group II an increase took place in the last 3 months. The values of the colour index were similarly lower than those in Group I (non-treated cases) in these periods. The values of the Hb, red cell count and the colour index for the Whole Series are also lower than those for Group I in all periods with the only exception of the red cell count in weeks 33–38, due to the fact that the Whole Series included all hypochromic anaemia cases in which the red cell count is less reduced than in other anaemic cases. The red cell values of the Whole Series show a rise from the 33rd to the 38th weeks, but it is significant that the red cell count remains stationary from the 39th to the 40th weeks owing to the inclusion of anaemic patients whose red cell count deteriorated in the last weeks before labour.

CRITERIA.

The mean colour index of non-treated cases in Group I attending for the first time was taken as the criterion for the normal physiological standard. In agreement with Price-Jones'[7] findings for parous healthy women, a colour index of 0.91–1.08 was taken as the standard for women not anaemic during the first 6 months of pregnancy. For the last 3 months the standard was the observed mean value in Group I (non-treated cases) for this period, i.e. 0.88 (75 examinations). Subdividing this period, the following were taken as mean colour indices: 27th to 32nd weeks, 0.93 (26 ex-

WHOLE SERIES—TABLE III.

ALL CASES, NON-TREATED AND TREATED WITH IRON OR PROTEIN OR LIVER IN PREGNANCY.

Pregnancy						Puerperium: Days postpartum		
Week	12–13	14–26	27–32	33–38	39–40	1–4	5–14	15–28
Red cell count	4.40	4.29	4.17	4.54	4.55	3.76	5.03	5.00
Haemoglobin (per cent)	79.8	75.8	73.5	72.3	74.0	60.0	68.0	78.0
Colour index	0.94	0.90	0.88	0.81	0.84	0.80	0.69	0.79
No. of examinations	(7)	(55)	(71)	(98)	(26)	(8)	(68)	(14)

MEAN VALUE OF ALL CASES AND ALL PERIODS OF PREGNANCY.

Red cell count	4.39 (257)
Haemoglobin	75.2 per cent (257)
Colour index	0.87 (257)

Total of cases in pregnancy, 94. Total of examinations, 257.

MEAN VALUE OF DIFFERENT PERIODS OF PREGNANCY AND PUERPERIUM.

		Red cell count	Haemoglobin	Colour index
Mean value of 1st 6 months	(62)	4.26	76.2 per cent	0.91
Mean value of last 3 months	(195)	4.36	73.2 ,,	0.83
Mean value, 39th–40th weeks	(26)	4.55	74.7 ,,	0.84
PUERPERIUM: Days postpartum.				
1–4	(8)	3.76	60.0 ,,	0.80
5–14	(68)	5.03	68.0 ,,	0.69
15–28	(14)	5.00	78.0 ,,	0.78

Total of cases in puerperium, 65. Total of examinations, 82.

aminations); 33rd to 38th weeks, 0.86 (36 examinations); 39th to 40th weeks, 0.88 (11 examinations). Cases with figures below these values were considered anaemic. The colour index in any one period of the last 3 months of pregnancy may occasionally be lower owing to the discrepancy between the course of the haemoglobin and the red cell count, but it was not taken as normal if it was maintained for more than 1 week and without a large and sudden increase in the red cell count. All mean values of the colour index of the Whole Series are lower than Group I (non-treated cases). The colour index in some forms of anaemia tends to unity, therefore the whole blood picture must be taken into consideration in relation to any given period of pregnancy.

PATHOLOGICAL DATA.

INCIDENCE OF ANAEMIA.

The 94 cases were divided into three categories according to the established standards:

	No.	Percentage of total cases
A. Non-anaemic cases, non-treated with iron, protein, or liver	43	45.8
B. Cases treated before first attendance and throughout pregnancy with routine administration of iron	7	7.4
C. Anaemic cases	44	46.8
		100.0

The high incidence of anaemia (46.8 per cent in a series of 94 cases) is comparable with that found by Davidson[8,9] in patients of similar ages and social classes (45 per cent in 3,500 cases).

Degree of Anaemia.

Out of 44 patients suffering from anaemia, 16 had a Hb level under 70 per cent more than once in pregnancy; 8 of these were patients suffering from hypochromic anaemia.

Besides the high incidence of anaemia, further progressive deterioration of the blood picture was observed in the last months of pregnancy, which agrees with Smallwood's[10] conclusion that occasionally severe and dangerous degrees of anaemia develop "during and as a result of pregnancy in the later months of pregnancy."

Classification of Anaemia.

The 44 cases of anaemia were classified according to their *blood picture* and their response to treatment.[11,12] The following types of anaemia were found which may have been unconditioned, or conditioned owing to insufficient intake[13] or defective assimilation of the diet or increased demands:

	No. of cases
1. Megalocytic protein deficiency anaemia	6
2. Hypochromic iron deficiency anaemia ...	23
3. Pernicious anaemia of pregnancy:	
(*a*) Hypochromic type—3 cases	
(*b*) Megalocytic hyperchromic type—4 cases	7
4. Vitamin C deficiency anaemia	8
	44

Those values were taken as representative for type and therapy which were found when the patient was first seen and before any treatment had been given (30 cases) or when anaemia developed after a normal beginning in the course of pregnancy (14 cases). In the first 6 months of pregnancy the blood picture follows that of anaemic parous women. Classification in the last 3 months of pregnancy was difficult because erythropoiesis was found even in hypochromic anaemic cases. As at least 3 features of the blood picture give evidence of the special type of anaemia a differential diagnosis is possible. In the last 3 month period of pregnancy erythropoiesis was found to be the dominating factor in the normal physiological non-anaemic cases of pregnancy. Although in anaemic pregnant women during this period the production of large fresh red cells is found and never quite disappears even in hypochromic anaemia in contrast to those in anaemic non-pregnant women, erythropoiesis is disturbed in a different degree determined by the type, and in an equal ratio to the gravity of the anaemia.

Variations in the Red Cell Count in the Last Three Months of Pregnancy.

Special variations in the red cell count during the last 3 months of pregnancy were observed only in the 7 cases of pernicious anaemia of pregnancy and in 5 out of the 8 cases of vitamin C deficiency anaemia. In the above-mentioned cases after a short initial increase in the number and size of the red cells at the beginning of the last 3 months of pregnancy a distinct decrease took place during the last weeks of pregnancy, and continued until labour. This decrease of the red cell count was found in no other cases than these 12, and in no other type of anaemia in the whole series of 94. The deterioration in the red cells in number and the reduction in their size was followed by post-partum haemorrhage. This connexion was found in 10 cases out of the 12 with a defective production of the red cells in the last weeks of pregnancy. In 2 cases this connexion could not be proved as blood tests could not be made at the appropriate corresponding time.

Postpartum Haemorrhage.

A loss of 10 ounces of free measured blood not soaked off in the towels or contained in the liquid was regarded as proof of haemorrhage. Out of 94 patients, 12 suffered from post-partum haemorrhage. Of these, 4, including 2 of Group I and 2 suffering from pernicious anaemia of pregnancy, had to be excluded either because they were not seen in the last 3 months before labour or until too long after it. Of the remaining 8 patients of measured post-labour haemorrhage, 5 had pernicious anaemia of pregnancy, and 3 vitamin C deficiency anaemia. Two further patients out of the 8 suffering from vitamin C deficiency anaemia, whose red cell count showed deterioration before labour, were assumed also to have suffered from post-labour haemorrhage, but the loss of free blood could not be measured. The red cell count of these 2 cases in puerperium was below the mean values for Group I in the same period. The loss in their haemoglobin level 9 and 13 days respectively after labour was still 11 and 17 points, which may be regarded as sufficient proof that post-labour haemorrhage had taken place.[4] In a second series of 20 pregnant women investigated later, 2 cases of vitamin C deficiency anaemia showed a similar progressive deterioration in the red cell count before labour, and a post-labour haemorrhage of 30 ounces and 16 ounces respectively. The connexion between defective production of red cells in number and size in the last weeks before labour and post-labour haemorrhage is, therefore, not a mere coincidence, but an observation of serious prognostic importance.

The Characteristics of Vitamin C Anaemia.

The 8 patients with vitamin C deficiency anaemia in the present series, and the 2 patients in the later series, suffered from latent scurvy, without obvious clinical symptoms other than breathlessness, lassitude, and malaise. The values of the blood picture were used as the standard for comparison with other anaemias. The blood picture was in agreement with that recognized by Mettier and Minot[14] for scurvy in adults. The red cells were more reduced than the Hb, the colour index tended to unity but was never above 1.0 as in megalocytic protein deficiency anaemia. The red cell count remained far below the mean value for Group I in the different corresponding periods. Microcytes and large red cells were observed at the beginning and during the course of the illness; megalocytes occurred rarely, if at all, in contrast to their frequent appearance in pernicious anaemia of pregnancy and in Addison's pernicious anaemia; some nucleated cells were also found. There was only slight central pallor in the large red cells, but no ringlike large cells. Microcytes were found, but not the dense-looking small microcytes found in pernicious anaemia of pregnant or of non-pregnant women. Anisocytosis was only moderate, but was seen more frequently than poikilocytosis. The failure of the blood to regenerate during the puerperium shows that the disturbing factor was still present after parturition and prevented a normal replenishment of the blood-forming elements. The reduction of the haemopoietic system chiefly affects the erythropoietic element. The values of the red cell count in the last 3 months of pregnancy and in puerperium even in patients in whom post-labour haemorrhage did not occur, were far below the normal values found in Group I, and also lower than in any other type of anaemia. The response to high dosages of iron (4 to 6 Pil. Ferr. Sulph.) given daily at the onset of the anaemia was delayed and inadequate, and the increase of reticulocytes was also delayed. Treatment with liver was ineffective.

Harris[15,16] and Zilva[17] refer to the absence of clinical signs of scurvy in patients proved by saturation tests to be suffering from vitamin C deficiency. Clinical signs of scurvy were not found even in patients kept experimentally for months on a diet[18] far below the League of Nations standard for vitamin C requirement of 30 mg. ascorbic acid daily.[22] Anaemia in scurvy is recognized by Mettier, Minot, and Townshend,[14] by Dunlop and Scarborough,[19] and by Isräls.[20] Some authors, however, challenge this view,[18] and attribute the anaemia to general under-nutrition.[21] It is considered that anaemia is not always associated with scurvy and that if it occurs in scurvy it is not due to loss of blood.[23] In the 8 cases of vitamin C deficiency anaemia neither large doses of iron nor of liver produced any regeneration of the blood during pregnancy or puerperium. The 2 patients of vitamin C deficiency anaemia in the second series received injections of Campolon 2 × 4 ccm. weekly from the beginning of the deterioration in the red cell count during the last weeks before labour. These liver injections neither prevented a further decrease in the number and size of the red cells, nor a heavy post-labour haemorrhage of 30 ounces and 16 ounces respectively. Injections of 250 mg. ascorbic acid 2–3 times daily had a sudden and dramatic effect in restoring normal haemopoiesis and producing a marked and rapid regeneration of the blood. In one of the two cases a relapse occurred after reduction of and full recovery after return to the former 3 dosages. The return to normal values began immediately after the injection. The increase in the reticulocytes also started after the injection and reached their peak after 20 hours in the one case seen at this period. All the blood-forming elements were restored after 12 days to the normal values for this period of the puerperium.

The improvement in the red cell count after the administration of ascorbic acid in the 2 cases of the second series cannot be attributed to improvement in nutrition as all patients in both series received the same general diet in the Fulham Maternity Home without the addition of food rich in vitamin C. Furthermore, Szent Györgi[24] *et al* have shown that pure vitamin C has a negative therapeutic action on haemorrhagic diseases of a non-scorbutic aetiology. It seems therefore permissible in the 2 cases of the second series to suggest the presence of a vitamin C deficiency anaemia dependent on continued lack of vitamin C in pregnancy and restored by a supply of the vitamin. All 8 patients of vitamin C deficiency anaemia in the first series did not receive any treatment with ascorbic acid before or after labour. The absence of improvement in the red cell count in these cases during the puerperium, whether there was post-labour haemorrhage or not, confirms once more the active presence of the aetiological factor or factors preventing normal haemopoiesis. The conformity of the blood picture in pregnancy between that given for scurvy in adults, the 2 cases in the second series and the 8 cases of vitamin C deficiency anaemia in the first series, suggests by analogy that the patients in the latter should probably be classified as suffering from vitamin C deficiency anaemia.

Obstetric History.

The different types of anaemia in the first series of 94 cases show widely different effects on the obstetric history of the mothers and on the children of the present pregnancy, hypochromic anaemia showing comparatively the most serious consequences. Seventeen out of 23 cases of children's diseases, dystrophy, low expectation of life, or rearing difficulties belonged to mothers with hypochromic anaemia. The 94 patients had 57 previous pregnancies, 36.8 per cent of which were a failure. There

were 7 miscarriages, 6 stillbirths, and 8 deaths of children. Four premature births are not included.

From the present pregnancies there were 6 stillbirths, 1 death (premature, died 4 months), 3 weak and prematurely born in Fulham Maternity Home and therefore included = 10.6 per cent.

Discussion.

A history of pre-existing anaemia was clearly defined only in 9 out of 94 patients of whom 5 showed anaemia in the present pregnancy. These 5 cases are not sufficient to justify the suggestion that anaemia in pregnancy is pre-determined. Against the assumption of Boycott[2] and Bethell[12] that anaemia is parallel to dilution of the blood stands the fact of the divergent non-parallel course of the haemoglobin and the red cell count in the last 3 months of pregnancy. Since the amount of the haemoglobin in the circulating blood is found to be the same throughout pregnancy,[5] whereas the red cell count is increased, the estimation of the haemoglobin alone is not sufficient as a criterion of the frequency of anaemia. Against the contention that there is a genuine physiological anaemia in pregnancy based on the falling Hb level, stands the fact that 43 patients not treated were found not to be anaemic, having a normal colour index throughout pregnancy. The higher activity of the bone marrow with the production of new large red cells in the last 3 months of pregnancy is the determining factor of the blood picture. Pregnancy in all its complexity has the tendency to rejuvenate tissues, of which the growth of the uterus and of the foetus proper are instances. Erythropoiesis found in all non-anaemic cases of Group I is therefore a physiological factor. It is besides a consistent factor as it was also found in anaemic cases, but on a smaller, varying, and differentiating scale.

From the blood picture observed it is inferred that in 44 cases anaemia was due to deficiency of iron, protein, or vitamin C which may have been unconditioned, or conditioned owing to insufficient intake or defective assimilation. Davidson (Aberdeen) and colleagues proved in their enquiry on over 3,500 individuals that a diet containing only 6 mg. iron (instead of 15–20 mg.) daily produced anaemia in women of child-bearing age, but not in men.[8,9] The requirement of vitamin C in pregnancy, especially in the latter half, is 60–100 mg. ascorbic acid—more than double that of non-pregnant women.[25,26] When the requirement is not met during gestation[22] the maternal organism becomes depleted for the benefit of the foetus at a time when erythropoiesis should be at its height. Thus pregnancy exerts its influence on, and intensifies such nutritional deficiency.

Conclusions.

(1) Forty-three cases out of a series of 94 pregnant women were found to be non-anaemic, having a normal colour index throughout pregnancy. The 44 anaemic cases were not of a predetermined character.

(2) The movement of the haemoglobin and the red cell count, both falling, is parallel in the first 6 months of pregnancy, but definitely non-parallel in the last 3 month period with a rising red cell count and a haemoglobin falling still further until the last 2 weeks before labour. The influence of hydraemia is therefore not the determining factor of erythropoiesis in pregnancy. The higher activity of the marrow bones is the dominating physiological and consistent factor of the blood in pregnancy.

(3) A decrease of the red cell count in number and in size before labour observed in two types of anaemia, i.e. pernicious anaemia of pregnancy and vitamin C deficiency anaemia, is a symptom of serious

prognostic importance likely to be followed by a post-partum haemorrhage.

(4) Eight cases of probable vitamin C deficiency anaemia of the present series, and 2 cases of vitamin C deficiency anaemia from a second series without any clinical evidence had the same blood picture as that found for scurvy in adults. The 2 patients in the second series could be identified as suffering from vitamin C deficiency anaemia because of their regeneration after high dosages of ascorbic acid and a relapse after reduction of those dosages.

(5) In view of the high incidence of anaemia in pregnancy in the 94 cases discussed and the tendency to further serious deterioration in the last 3 months of pregnancy, it would be advisable to make a full blood test in each 3 months period of pregnancy and also in the last weeks before labour.

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Neonatal Mortality

BY

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This paper formed the basis of a discussion on neonatal mortality in the Ulster Medical Society in March, 1943.

It analyses the neonatal mortality of 6,314 babies born in the Royal Maternity Hospital, Belfast (ALLEN), 943 babies attended in private practice (MACAFEE), and the findings at the postmortem examinations of 125 babies who died during the neonatal period (BIGGART).

F. M. B. ALLEN.

THE importance of improving the welfare of infants and the health of children must be placed in the forefront of the better health services to which we all look forward. The falling birth-rate makes the conservation of child life of genuine importance to the nation.

In Northern Ireland, in 1939, 260 babies died on the 1st day of life; 302 in the next 6 days, and 237 in the 2nd, 3rd, and 4th weeks—a total of 799 babies lost in the neonatal period. In England and Wales it is estimated that in any one year 17,000 babies die during the same period.

It is obvious that a field of fruitful activity is open for those who are anxious to achieve a reduction in this wastage of human life. That a reduction is possible is shown in Chicago where a neonatal death-rate of 53.4 in 1930 was reduced to 28.8 in 1940. During the same period in the State of Illinois the rate was reduced from 56 to 35.1.[1] This was achieved by providing an extensive paediatric service supervized by an authority interested in infant welfare and by the inspiration of an enthusiastic paediatrician.

The factors which influence the survival of the infant at various stages of its development have been summarized in a memorandum.[2]

TABLE I.[3]
Neonatal Mortality Rates.

	England and Wales*	Scotland	Northern Ireland
1939	28.1	36.6	31.7
1940	28.83	37.3	34.8
1941	28.52	39.9	33.8
1942	26.99	35.1	34.9*

* Provisional rates.

(1) HEREDITY.

Those interested in eugenics stress the importance of heredity. Every family doctor recognizes the truth of the saying that "breeding always tells" and sees it exemplified time and again in the nursery of the newborn.

(2) CONGENITAL MALFORMATIONS.

The importance of this factor as a cause of stillbirth and neonatal death will be noted later in this paper. As these malformations have their origin in embryonic life any possible reduction in the incidence of this factor is doubtful until research throws more light upon their origin.

(3) Toxaemic Factors.

Syphilis: Pre-eclamptic toxaemia: Diabetes.

(a) *Syphilis.* Routine Wassermann tests in the Antenatal Department of the Royal Maternity Hospital for some years revealed an incidence of more than 1 per cent of unsuspected syphilis. The war-time increase in the incidence of syphilis calls for more attention to the possibility of imperfectly-treated maternal infection. Biggart's figures, however, would suggest that syphilis, as a cause of death in the neonatal period, is not an important factor.

(b) *Pre-eclamptic toxaemia.* There is a possibility that pre-eclamptic toxaemia may adversely influence the welfare of the infant in intra-uterine and neonatal life. A dogmatic statement on this cannot be made in the light of our present knowledge, but personal observation lends support to this view. This type of toxaemia does, however, increase the number of premature infants and a large percentage of the mothers of these babies have difficulty with breast feeding.

(c) *Diabetes.* This is an important factor in some cases of intra-uterine death and there is a high mortality among the babies of diabetic mothers in the 1st week of life.

(4) The Premature and Immature Infant.

For the purpose of uniformity all infants weighing less than 5½ pounds at birth are regarded as immature, irrespective of their estimated maturity. The well-recognized relation of immaturity to neonatal deaths is strongly borne out in this paper.

(5) Natal Conditions.

These conditions are mainly the concern of the obstetrician and include birth trauma, respiratory distress, etc.

(6) Postnatal Conditions.

Lack of adequate skill, especially for premature babies, inability or lack of desire to breast feed and the failure to protect the infant against infection are important factors in accounting for neonatal mortality. From a paediatric standpoint factors influencing lactation are imperfectly understood and further information is urgently desirable.

Table II.

Neonatal Deaths in Royal Maternity Hospital, Belfast.

Mature babies			
	Born	Died	Mortality-rate
1939	1198	14	1.17
1940	1399	20	1.43
1941	1538	11	0.72
1942	1516	23	1.51
Total	5651	68	1.2

Immature babies			
	Born	Died	Mortality-rate
1939	149	27	18.1
1940	131	38	29.0
1941	178	29	16.3
1942	205	49	23.9
Total	663	143	21.5

A detailed analysis of the deaths during the 4 years 1939–42 has been carried out. During this time 6,314 babies were admitted to hospital nurseries:

5,651 were mature and 68 died (mortality 1.2 per cent).

663 were immature and 143 died (mortality 21.5 per cent).

A postmortem examination was not made in all cases; in a few because the reason for death was obvious either from the clinical history or the physical examination (spina bifida, intracranial haemorrhage,

etc.); in others the provisional diagnosis was confirmed at necropsy; in the remainder the explanation of death was only revealed in the postmortem room. In passing it should be noted that in many cases the recorded cause of infantile death is inaccurate and unconfirmed by postmortem examination (Spence, Miller[3]). Tables III and IV, however, present a picture of neonatal pathology as experienced in the Royal Maternity Hospital and show the importance of developing this field of pathological research.

TABLE III.

The cause of death in 68 mature babies was:

Congenital malformations	27
Intracranial haemorrhage	15
Infection	9
Respiratory distress	5
Erythroblastosis foetalis	3
Haemorrhage neonatorum	1
Mongolism	1
Renal thrombosis	1
Undetermined	6

TABLE IV.

The cause of death in 143 immature (premature) infants was:

Prematurity	76
Infection	15
Congenital malformations	16
Intracranial haemorrhage	11
Respiratory distress	15
Mongolism	2
Erythroblastosis foetalis	1
Haemorrhage neonatorum	1
Undetermined	6

The large number of congenital malformations accounting for neonatal deaths was somewhat surprising. Out of 211 (i.e. total neonatal) deaths 43 (20.4 per cent) were attributable to this cause (39.7 per cent in mature babies and 11.2 per cent in immatures).

This is not the occasion to record the number of stillbirths associated with congenital malformation; but it is possible that a similarly high figure would be found.

TABLE V.

Nature of Congenital Malformation:

Spina bifida	19
Meningocele	2
Hare lip, cleft palate	2
Alimentary	8
(Including oesophageal atresia, duodenal occlusion, imperforate anus.)	
Hernia of diaphragm	3
Ectopic vesicae	1
Congenital heart disease	8
(Including patent ventricular septum, patent foramen ovale and coarctation of aorta.)	

The outstanding observation as regards deaths is that prematurity accounted for 76 of the 143 deaths among immature infants (53.1 per cent) and for 76 out of 211 total neonatal deaths (36.01 per cent).

TABLE VI.

Deaths of 143 Immature Infants Related to Age at Death.

Survival	1	2	3	4	5	6	7	8	9	10	11	12 or more days
No. of deaths	73	18	4	5	7	4	2	2	2	0	0	26

This indicates clearly that the first two days, and especially the first day, are the most critical as regards survival and if an immature infant can be kept alive for the first 48 hours its prospects are greatly improved. The 26 deaths occurring on the 12th or subsequent days are mainly those due to congenital malformations and neonatal infection.

THE ROLE OF INFECTION.

More than 11 per cent of the deaths were due to neonatal infection (9 among mature and 15 in immature babies). The role of infection as a cause of death in the newborn is not appreciated as it should be; this is the more regrettable since it is largely preventable. There is a tendency sometimes to attribute to innocent causes deaths which postmortem examination reveals as being

due to neonatal sepsis. An asphyxiated baby may be presumed to have died of asphyxia neonatorum when it really had neonatal pneumonia; or of severe icterus when septic cholangitis was present.

Natal Conditions.

Birth trauma (usually intracranial haemorrhage) and respiratory distress accounted for 46 out of 211 deaths (21.8 per cent). The term respiratory distress is used in preference to asphyxia neonatorum and includes asphyxia from central causes, respiratory obstruction and atelectasis, injudicious premedication and rarer causes incidental to birth, such as interference with the umbilical vessels by knots and twists.

It is admittedly difficult to account for neonatal asphyxia in all cases; the factors to be considered are cerebral damage, neonatal pneumonia or atelectasis and congenital heart disease. Necropsies have been particularly useful in elucidating the cause of death because physical examination of the newborn infant is never easy.

The Reduction of Neonatal Mortality.

Research in the future may provide information concerning the occurrence of congenital malformations. Obstetricians are aware of the deaths which are directly associated with birth. Better obstetric practice should reduce the incidence of birth trauma, particularly intracranial haemorrhage, and in some cases prevent respiratory distress. An attempt should be made to advance the maturity of the infant as far as possible without prejudicing the safety or welfare of the mother.

Special attention has been given to the care of immature and premature infants in the Royal Maternity Hospital in an attempt to lower the mortality among them which at one time was over 40 per cent. To achieve the reduction certain principles were observed and a routine was established. Especial care is taken in the labour room to avoid chilling of the infant so that abnormally low body temperatures do not occur. A specially trained nursery staff was appointed, the aim being that the nurse in charge should be prompt to recognize the failure to respond to treatment and be competent to apply the special measures necessary. The nursing staff is under the charge of a specially-trained Sister, and it is noted that ' black periods ' in the records usually coincide with a change in staff. A special nursery is set apart for immature infants, which can be kept at a higher temperature of about 78°F.; here additional moisture is added to the atmosphere so that a humidity of 60 to 65 is attained. This maintains body warmth and avoids excessive fluid loss. For the smaller babies oesophageal feeding is almost the rule; for those who are able to nurse from a bottle for a short time before tiring a modified feeding bottle, preferably of the Belcroy* type, is used. Breast milk is regarded as the most satisfactory means of feeding, various substitutes having been thoroughly tried and proved inferior. A "pool" of surplus breast milk from mothers in the hospital is available for the feeding of immature infants. Each infant is kept warm in a simple type of electrically-heated incubator.[4] The baby is wrapped in sterile gamgee and cleanliness achieved by the application of sterile olive oil. Visitors are not allowed into the nursery and those whose duty necessitates their admission must wear an effective mask over the mouth and nose. Babies who show any indication of infection are immediately segregated.

Neonatal infection is next in importance to immaturity as a cause of death and in itself exceeds in number the deaths due to

* Bell and Croydon, London.

or associated with the delivery of the baby. Therefore any steps which can be taken to overcome and avoid an acquired condition which is so fatal are surely worth while. In hospital the rule is to segregate immediately a pyrexial infant and to ascertain the cause —the upper air passages, the respiratory tract and the alimentary tract are the most common sites. Local infection of the umbilicus is sometimes associated with infective cholangitis; B. coli pyelonephritis (often with malformation of the genito-urinary organs) and, on two occasions, B. coli meningitis have been found. Cutaneous infections are troublesome and demand isolation, but as a rule they respond to local treatment. In an epidemic of pemphigus neonatorum some years ago 54 cases occurred with 1 death, the cause of the epidemic being eventually traced to a laundry maid, engaged in folding napkins, who was infected with impetigo. It has been found that the use of an effective mask by all those whose duty it is to enter the nurseries assists in reducing neonatal infection. It must be realized that the portal of entry of the infective agent is very often the respiratory tract and that adults may project infected droplets by talking, coughing or even breathing near an infant. Neonatal infection in private practice almost always occurs when no such precautions are taken. In the Royal Maternity Hospital the "mask rule" was relaxed, but after 2 weeks the nursing staff asked that it be reimposed because 7 babies developed pneumonia and of these 2 died. The rule was again relaxed (except for the premature nursery) for 18 months from 1940, nurses being placed on their honour to report if they had any respiratory infection. During these 18 months of the four-year period under review, 5 out of 6 fatal cases of pneumonia occurred, 5 out of 8 of gastroenteritis and 1 out of 2 of septicaemia.

Paediatricians are convinced that breast feeding is in every way superior to artificial milk preparations. Figures are available showing that morbidity is higher among bottle fed babies and that the mortality is several times higher. It is notable that apart from the occurrence of conditions such as pyloric stenosis, mental defect, etc., attendance of infants at the outpatient departments of children's hospitals, and in private practice, is almost confined to those fed on breast milk substitutes. It is tragic to record that doctors and nurses are often among those who advocate artificial in preference to breast feeding. In many cases the reason would appear to be the lack of appreciation of the necessity for natural feeding and knowledge of its proper management. It must be realized that the proper teaching of students, nurses and doctors should be undertaken by paediatricians who are familiar with the difficulties encountered. Until adequate instruction is widely given public health campaigns will not achieve a full measure of success. Weaning sometimes takes place in the 2nd or 3rd week of lactation owing to a temporary failure when the mother resumes her household duties. It can often be avoided by resorting to supplementary feeding for a short time pending restoration of the natural supply. Again, the young, less experienced doctor, filled with enthusiasm for the practice of breast feeding encounters at times the ridicule of the older generation, and is unable to contend with the misguided mother who is influenced by commercial literature or the advice (and even threats) of her friends, parents, or "in-laws."

Infant welfare centres should play a large part in instructing and advising mothers, especially those who are inexperienced. That a service of this kind is welcomed has been proved at the Royal Maternity Hospital where the mother is taught to feed,

bath and dress her own baby before discharge, and where there is an infant clinic for 'well' babies up to 6 months. There is, unfortunately, no assurance that the staffs of all welfare centres have sufficient knowledge or have gained experience by residence in or attendance at a maternity and a children's hospital. It is essential that the Infant Welfare Medical Officer should have held the post of Resident Medical Officer in a Children's hospital and a Maternity hospital (preferably teaching) for at least 12 months.

There is an increasing appreciation of the importance of the maternal diet in relation to the success of lactation and the infant's welfare. A clinical assessment of the mother's health and prenatal diet suggests that the better they are, the more satisfactory will be the infant's progress. Evidence in support of this is accumulating and should soon be available. It has been stated that "a well-balanced diet of 2,500 calories for the last 3 or 4 months of pregnancy and during the early lactating period will result in fewer stillbirths, less delicate newborn babies and better maternal lactation. Even the provision of one meal daily of 900 calories is of undoubted value."[2]

SUMMARY.

(1) An analysis of neonatal deaths over a four-year period is presented. It emphasizes the proportion of deaths due to congenital malformations incompatible with life, and of the role played by prematurity as the chief cause of death among immature infants.

(2) Steps to reduce neonatal mortality include better care of the immature infant, avoidance of neonatal infection, maintenance of breast feeding, and instruction on the management of natural feeding.

(3) Obstetricians should delay the induction of premature labour as much as possible (without prejudice to the mother's welfare) so that the infant may be as mature as possible at birth.

(4) There should be increased and improved welfare services, associated with better instruction of the undergraduate, the family doctor and maternity nurses, on infant management and hygiene.

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C. H. G. MACAFEE.

This communication deals with what one might regard as a selected group of the population, namely private patients, in contradistinction to Dr. Allen's paper, which gives the figures of a large maternity hospital receiving patients from varied social strata.

Table I gives the gross figures for the whole group.

TABLE I.

Patients	928
Babies	943
Stillbirths	20
Neonatal deaths	16
Death-rate per 1,000 live births	17.34
N.I. Same period (average)	33.5

In going through these figures I was struck with the difference between two groups, viz. the figures for the period 1926–37, and those for the period 1938–42.

I understand from the statisticians that one cannot arrive at any conclusions from

such a small series. Nevertheless if the causes of the stillbirths and neonatal deaths in these two groups are analysed some interesting points arise.

TABLE II.

	1926 to 1937	1938 to 1942
Patients	504	424
Babies	513	430
Stillbirths	15	5
Neonatal deaths	12	4
Death-rate per 1,000 live births	24.04	9.48
N.I. same period (average) ...	33	34

TABLE III.

Stillbirths

	1926–37	1938–42
Death in labour ...	6	0
Macerated foetus* ...	4	1
Anencephaly	3	2
Albuminuria and placental infarct.	1	0
Entanglement of cord	1	0
Second twin	0	1
Cerebral haemorrhage	0	1
	15	5

Neonatal deaths

	1926–37	1938–42
Prematurity	6	0
Congenital abnormalities	3	1
Asphyxia	2	0
Cerebral haemorrhage	1	1
Enteritis	0	1
Acute streptococcal infection ...	0	1
	12	4

Summary:—		per cent
Pre-eclamptic toxaemia (resulting in prematurity or maceration)	11	30.5
* (One case of maceration not associated with pre-eclamptic toxaemia).		
Congenital abnormalities ...	9	25.0
Birth trauma	3	8.3
Neonatal infection	2	5.5

The occurrence of 6 deaths in labour in the first group seems very high. These deaths, however, resulted from the following conditions:—

Placenta praevia	2
Prematurity (32 weeks)	2
Breech	1
Unexplained	1

The occurrence of 9 cases of congenital abnormalities (accounting for 25 per cent of the total) shows an incidence which corresponds closely to that of Dr. Allen's series and is high when compared with other areas. For example, congenital abnormalities only accounted for 12 per cent of the stillbirths and neonatal deaths in the Rotunda in 1940–41.

Of the 3 cases of cerebral haemorrhage, one was due to an error of judgment for which I was partly responsible, one occurred in an easy spontaneous delivery and in the third case the delivery was accelerated on account of a cord prolapsing while the head was being rotated from the occipito-posterior position. The rapid delivery almost certainly caused the tentorial tear.

The 2 cases of asphyxia resulted from the babies being left unattended in their cots sucking bottles of water.

Of the 2 cases of neonatal infection one was acquired from the child's grandmother who insisted on visiting the infant while recovering from an acute streptococcal throat.

Of the 27 stillbirths and neonatal deaths in the first group pre-eclamptic toxaemia resulting in a macerated or premature infant accounted for 11 deaths. In this group the incidence of pre-eclamptic toxaemia necessitating induction of labour was 12.9 per cent. In the second group prematurity does not figure as a cause of death. The incidence of pre-eclamptic toxaemia necessitating induction of labour in this second group was only 4.9 per cent.

and in all but one case the induction of labour was carried out in or after the 36th week.

Are there any factors which may have influenced the better results obtained in the second group?

While it is impossible to be dogmatic I think there are at least three factors which have had an influence. In the second group I certainly paid more attention to the patient's diet during pregnancy, and in all cases this was supplemented by iron, calcium and vitamin products. In the case of pre-eclamptic patients, apart from rest in bed, the rigorous dietetic treatment advised in the past has not been enforced.

There is another factor which, I think, is most important. This is, that unofficially, pregnancy has become a notifiable condition. At the present it is a great advantage to be an expectant mother. She receives priority eggs, milk, clothing coupons, from the earliest days of her pregnancy, and to obtain these she not only acquaints her doctor but unblushingly notifies her milkman, grocer and chemist. This results in the patient receiving antenatal advice and treatment many weeks before the average patient usually saw a doctor in the past.

That there are many advantages in this is undoubted and these advantages have been borne out by a paper recently published by Nixon[1] on "Diet in Pregnancy." He includes in his paper a table, part of which is reproduced.

The comparison of my two groups reveals the third factor. I have noted that in the second group of cases prematurity does not figure as a cause of death. This does not mean that there were no premature babies in the second group. The great majority were born in two private institutions, in one of which the facilities for the care and nursing of premature infants are the best available in this area.

In the other the Matron takes personal care of all premature infants and is most successful in their management. The figures therefore represent the results of careful antenatal supervision, with attention and additions to the diet, the minimal amount of trauma at delivery and efficient nursing of the infant afterwards.

I think that all will agree that the results in any series of cases depend not only on the medical attendant but also on the intelligence and co-operation of the mother (especially in cases of toxaemia), the surroundings in which the baby is born and, most important, the nursing facilities available.

If my conclusions are correct they point to certain ideals for the future.

First, that adequate antenatal supervision not only entails the recognition and avoidance of obstetrical complications but also supervising and supplementing the patient's diet. Where financial circumstances do not permit of this diet being adequate it should be supplemented by the State, and in return for this assistance the

TABLE IV.

	Stillbirths and neonatal deaths (calculated per 1,000 total births)
1. Cases receiving special foods	59
2. Cases not receiving special foods but attending antenatal clinics	71
3. Cases not receiving special foods and not attending antenatal clinics	92

patient should be willing to be supervised from the earliest days of pregnancy.

Secondly, that adequate provision should be made for every class of patient in efficiently organized and supervised institutions where premature infants can be nursed. At the present time in the British Isles the scarcity of accommodation for maternity cases is a serious problem for all expectant mothers and those who attend them, and provision for the premature infants is so limited that many cannot receive the best possible treatment.

The question of pre-medication in labour being a danger to the infant has been raised. While the indiscriminate and injudicious use of sedatives in labour is dangerous, I cannot agree that one has any justification in denying the mother relief from pain on account of, what I have personally found to be, a problematical risk to the baby. My second group of cases was the more heavily and generally premedicated of the two and yet the results, as far as the baby is concerned, were better. I have had experience of over 500 cases in which the combination of barbiturates and large doses of scopolamine have been used and in one only have I ever thought that the pre-medication had any influence on the foetal mortality. This was in one unexplained death occurring late in the 1st stage of labour.

I am satisfied, however, that the use of morphia in any form during labour is dangerous to the baby as it is liable to increase the risk of respiratory difficulties.

Co-operation between a skilled anaesthetist and the obstetrician at any delivery is an important factor in the establishment of respiration and the prevention of asphyxia.

Professor Biggart's figures show that of the postmortem examinations performed on stillborn babies or those dying shortly after birth, 18 per cent show some congenital abnormality which accounts for the foetal death. This figure does not include the obvious abnormalities such as hydrocephalus and anencephalus.

In Dr. Allen's series foetal abnormalities, obvious or revealed at postmortem, as a cause of death, accounted for 20.4 per cent, and in my own series the figure was 25 per cent.

It seems, therefore, that an unexplained foetal death in any case in which sedatives have been used is more likely to be due to some congenital abnormality, e.g., cardiac or intestinal, rather than to the sedative used.

I feel that it is unfair to hold the bogey of "It is dangerous for the baby" over the mother in order to avoid the extra trouble entailed for both doctor and nurse when any form of sedative is used.

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J. H. BIGGART.

The findings at postmortem in 125 consecutive cases are tabulated in Table I. While the series is too small to justify statistical analysis the high ratio of congenital abnormalities is worthy of note (18 per cent). The present material contains none of those apparent malformations of the central nervous system such as hydrocephalus, anencephalus, meningoceles, etc. It consists entirely of malformations of the viscera which of themselves initiated the series of events which resulted in death of the child. Congenital malformations of the heart (10), atresia of the oesophagus (2), oesophageal-tracheal fistula, diverticula of the colon with perforation, volvulus of abnormal intestines, large diaphragmatic hernias producing

atelectasis, have all been encountered. Many of these conditions are difficult to diagnose in the young child, and their presence can only be detected at postmortem.

rupture of the ascending colon apparently of traumatic origin. The case of mediastinitis was secondary to an acute oesophagitis. It is of some interest that in the five-year period during which these cases

TABLE I.

TOTAL POSTMORTEMS 125.

Tabulated Causes of Death.

Postmortem findings	No.	1st week	2nd week	3rd week	4th week
Congenital abnormalities	23	14	6	3	–
Intracranial haemorrhage	20	20	–	–	–
Prematurity	13	10	1	1	1
Atelectasis	9	9	–	–	–
Infections (36).					
Pneumonia	15	4	3	6	2
Septicaemia	8	–	5	2	1
Peritonitis	3	1	1	–	1
Enteritis	4	–	–	3	1
Meningitis	2	1	–	1	–
Laryngitis (non-diphtheritic)	1	–	–	1	–
Pyelonephritis	1	–	–	1	–
Empyema	1	1	–	–	–
Mediastinitis	1	–	1	–	–
Erythroblastosis	7	7	–	–	–
Renal venous thrombosis	6	–	4	2	–
Oedema of lungs	3	3	–	–	–
Malnutrition	2	–	–	–	2
Cerebral venous thrombosis ...	2	–	1	1	–
Jaundice	1	1	–	–	–
Trauma	1	–	–	–	1
Haemorrhage	2	1	–	1	–
Total	125	72	22	22	9

Intracranial haemorrhage, chiefly due to tentorial tears, accounts for 16 per cent of the total deaths. One case of intraventricular haemorrhage and one of pontine haemorrhage were seen.

Infective lesions constitute 29 per cent of the total, almost half being infections of the respiratory system. Those dying in the first week were due to aspiration pneumonia (4), B. coli meningitis, B. coli empyema, and faecal peritonitis with

were collected there has not been a single case of congenital syphilis dying in the first 4 weeks of post-natal life.

Cerebral marantic thrombosis has been seen in 2 cases. Even more interesting are the 6 cases of bilateral thrombosis of the renal veins. The occlusion of these veins was the only finding at postmortem. Evidence of sepsis was not obtained, and it would appear that they must be considered of marantic origin.

Erythroblastosis and Congenital Syphilis in a Newborn Infant

BY

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AND

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THE chance association of erythroblastosis and congenital syphilis in an infant recently born in the Simpson Maternity Hospital aroused much interest, not only because of its rarity, but also because of the difficulty of differentiating these two diseases which have so many features in common. Little was known about erythroblastosis until the last decade and most of the more severe cases with stillbirth or miscarriage were diagnosed as congenital syphilis. A history of congenital jaundice, hydrops or intrauterine death, in successive pregnancies, was regarded as strong confirmation of the diagnosis of congenital syphilis, and women with such an obstetrical history were frequently given prolonged courses of anti-syphilitic treatment in spite of negative serological tests for syphilis. Extensive research and increased publicity during recent years have greatly increased our knowledge of erythroblastosis and more general recognition has shown the disease to be commoner than congenital syphilis.

Recently, one of us (Henderson[1]), in a paper entitled "Erythroblastosis or Congenital Syphilis?" analysed the principal clinical and pathological features of these two diseases and discussed the differential diagnosis. The valuable serological test for erythroblastosis was not mentioned as it had not passed the experimental stage when this paper was written. The test is dependent on the development in the mother of anti-Rh agglutinins, Rh (rhesus) being a factor in the foetal erythrocytes which is inherited from the father. The test is proving of great value in the differential diagnosis of erythroblastosis when expectant mothers give a history of recurrent miscarriage and/or intrauterine death. In the case about to be recorded the mother's Wassermann and Kahn tests for syphilis and the serological test for erythroblastosis were all positive. The hydropic infant died half an hour after birth. The cord-blood taken at birth gave a positive Wassermann reaction. A postmortem examination seemed to confirm the provisional diagnosis of congenital syphilis, but the histological features resembled those of erythroblastosis much more closely than those of syphilis.

CASE RECORD

Baby U; female; born January 21st, 1943; first child; illegitimate.

History of mother. Age 21 years. Four months after the last menstrual period she complained of a sore on the left side of the vulva. A few weeks later she noticed a rash on the abdomen and thighs which disappeared after 2 weeks. It returned a month later and became so irritable that she could not sleep. A few weeks later she went to a doctor who observed a rash on the back and abdomen and diagnosed secondary syphilis. She was admitted to the venereal diseases ward of the maternity hospital on January 6th, 1943. Examination on admission

showed what appeared to be the primary sore on the left side of the clitoris and condylomata lata on the vulva. A fading roseolar rash was noticed on the skin of the abdomen and thighs and there were small condylomata under the left breast. One rubbery gland was palpated in the left groin and a few small ones in the right groin. The spirochaeta pallida was demonstrated in serum from a labial condyloma and also from a submammary condyloma. Wassermann reaction ++, Kahn reaction +++. Gonococcal fixation test −. One week after the commencement of treatment the rash had entirely faded and the condylomata had almost healed. About a month later, after histological examination of the foetal tissues, the blood was investigated for evidence of erythroblastosis. The erythrocytes were found to be Rh negative, and anti-Rh agglutinins were demonstrated in the serum (++).

History of infant. A female infant was born spontaneously 15 days after admission of the mother to hospital. It weighed 5 pounds 15 ounces. It showed pronounced generalized oedema with great ascitic distension of the abdomen. Was sermann reaction of cord-blood +. The placenta weighed 1 pound 9 ounces and was abnormally thick; it had a rather pale colour and showed multiple small infarcts. The infant died half an hour after birth.

Postmortem Examination of Infant.

General appearances. A female infant weighing 5 pounds 15 ounces. There was a considerable amount of generalized subcutaneous oedema. The abdomen was greatly distended and the umbilicus protruded.

Head. There was no intracranial haemorrhage. The leptomeninges were extremely congested.

Thorax. Pleural sacs were healthy. Pericardial sac contained a slight excess of clear fluid. There were no subserous haemorrhages.

Lungs were very poorly expanded and contained only a little air in the anterior borders. Otherwise they showed nothing of interest.

Heart was a little dilated and was rather large in proportion to the size of the body.

Thymus gland was small.

Abdomen. Peritoneal sac contained a very large quantity of clear yellow fluid.

Alimentary tract showed nothing to note.

Liver was a little enlarged, firmer than usual and of fairly normal colour but a little browner than usual. It was possibly very slightly tough to cut. The outer surface was smooth but showed a very slight tendency to granularity when put on the stretch.

Spleen was greatly enlarged, being fully 3½ inches in length. It was of about the usual consistence and colour and not tough. The Malpighian bodies were invisible.

Kidneys and suprarenals showed nothing of interest.

Bones. The bones at both knees were examined. Both fibulae, which were cut into, showed a relatively bright yellow line at the epiphyses which was very suggestive of syphilitic infiltration.

Histology.

Liver. (Figs. 1 and 2.) Haemopoietic activity was very great. Some of the foci were mainly or entirely normoblastic, but many were composed of large primitive erythroblasts. This feature was quite characteristic of erythroblastosis. The centre of every hepatic lobule showed an area of necrosis in which haemorrhage had occurred and all the liver cells had either disappeared or lost all nuclear staining. In the outer zones the liver cells were healthier, but they showed separation in the columns, while individual cells were distorted and variable in size, some being atrophied, some containing several nuclei. In the peripheral zones there was a slight but quite definite pericellular fibrosis, which was not detectable in the necrotic central zones. The liver cells contained a considerable amount of haemosiderin. Spirochaetes were not found after prolonged search of sections stained by Dobell's method.

Spleen. The most striking feature was active haemopoiesis, with many primitive erythroblasts. Fibrosis was not evident. The Malpighian bodies were small. These changes were characteristic of erythroblastosis and it was doubtful whether any syphilitic changes were present. No spirochaetes were found in sections stained by Dobell's method.

Blood. Nucleated red cells were numerous. There were many normoblasts and numerous more primitive cells. Anisocytosis was a feature.

Bone (fibula). Syphilitic infiltration was not discernible in the region of the ossifying junction. The marrow was actively erythroblastic.

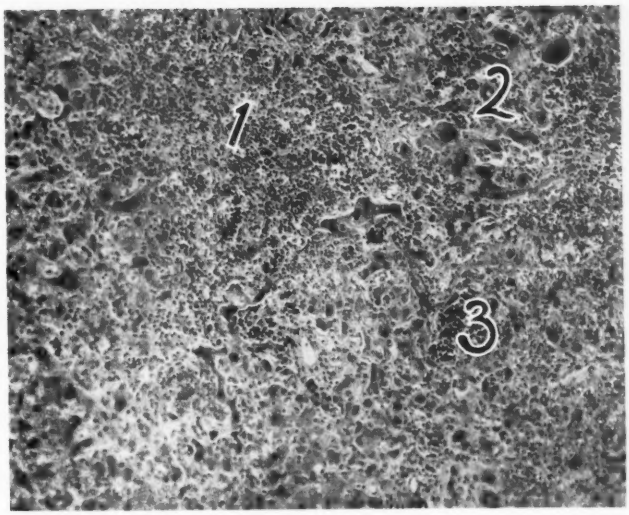

FIG. 1.

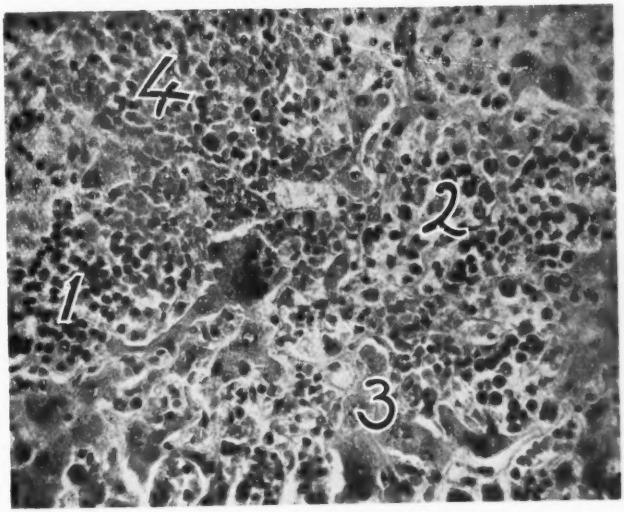

FIG. 2.

H. & M.

Lung. Nothing abnormal was seen.

Placenta. The only remarkable features were slight stromal oedema and an excessive number of nucleated red cells in the vessels. Spirochaetes were not found in sections stained by Dobell's method.

Comment.

The evidence of erythroblastosis in this remarkable case is clear. Active haemopoiesis of a primitive type in the liver and spleen, central necrosis of the liver lobules, haemosiderin in the liver cells and erythroblastaemia are all characteristic features. The pronounced generalized oedema with great ascitic distension of the abdomen, observed at birth, is more suggestive of erythroblastosis than of congenital syphilis, and the presence of anti-Rh agglutinins in the mother's serum strongly supports a diagnosis of erythroblastosis.

The evidence of congenital syphilis is less definite. The histological evidence, unlike that of erythroblastosis, is indefinite. The fibrosis of the liver, unrelated to the necrosis, and the distorsion and atrophy of liver cells in the peripheral (surviving) parts of the lobules, were suggestive features, but in the presence of erythroblastosis as an alternative cause of fibrosis and in view of the failure to demonstrate spirochaetes, perhaps not conclusive. The failure to demonstrate spirochaetes is not vitally important, as they may be much scantier in viable than in macerated tissues and difficult to find in stained sections. They can seldom be demonstrated in the placenta in any circumstances. Infection of an infant's tissues with the spirochaeta pallida can be presumed when the mother is suffering from untreated secondary syphilis of several months' duration, and the infant's blood shows a positive Wassermann reaction, as in this case. The macroscopical postmortem feature that most strongly suggested syphilis was the slight toughness of the liver, but this feature does not eliminate erythroblastosis, for a diffuse fibrosis is occasionally found in that disease also.

It is not surprising that a provisional clinical diagnosis of congenital syphilis was made in this case, in view of the florid untreated syphilis of several months' duration in the mother. In these circumstances, the hydropic state of the infant observed at birth and the very large spleen demonstrated at autopsy, though more characteristic of erythroblastosis than of congenital syphilis, did not create serious consideration of the former disease. Erythroblastosis was not suspected until the tissues were examined microscopically. The unmistakable evidence of erythroblastosis which this revealed led to the examination of the mother's serum for the presence of anti-Rh agglutinins. The demonstration of these antibodies confirmed the existence of erythroblastosis. If the presence of congenital syphilis should be regarded as unproven, the case loses little of its interest, for the clinical and macroscopic diagnosis of congenital syphilis appeared almost indisputable.

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LEGENDS

Fig. 1. (1) Liver showing areas of necrosis and haemorrhage, (2) fine pericellular fibrosis, distortion of liver cells, and (3) haemopoietic foci.

Stain: H. & E. × 100.

Fig. 2. (1) Liver showing foci of normoblasts, and (2) primitive erythroblasts, (3) liver cells containing haemosiderin granules and (4) margin of a necrotic area.

Stain: H. & E. × 100.

The Nomenclature of Hormone-Producing Tumours of the Ovary

BY

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SINCE gonadal hormones were first obtainable in pure form, our comprehension of them has extended widely and advanced with great speed. The newer knowledge shows that some of the current terms of pathology have questionable foundations and may perhaps convey misleading implications. Among such terms are those applied to ovarian tumours which disturb the manifestations of sex, namely 'arrhenoblastomata' and 'granulosa-cell tumours.'

ARRHENOBLASTOMA.

The strict meaning of this name is, it may be supposed, a tumour arising from a masculine rudiment. Robert Meyer,[1] to whose careful investigation of these tumours we shall always be indebted, states "the fact that they can produce male characteristics, in my opinion, does not of itself furnish sufficient ground to call them arrhenoblastomas." Later he adds that "another reason for calling these tumours arrhenoblastoma of the ovary is based on the theory that, whether or not they produce male characteristics, they always arise from definitely male-directed cells," and "the resulting masculinization confirms the theory of the origin of this new growth." Here we have two possible fallacies, namely: (1) that these tumours always arise from 'male-directed' cells, by which Meyer seems to imply male vestigial cells, and (2) that the resulting masculinization confirms this assumption. Apparently it was thought that because the tumour caused masculinization it must, therefore, have been derived from a persistent male rudiment. However, as Meyer himself remarks, tumours which show the typical histological picture, namely the tubular adenomas, do not always cause masculinization or defeminization. Moreover Novak and Long[2] have pointed out that ovarian tumours of diverse histological types may cause hairiness and other manifestations of virilism, although if sections from all parts of the tumour be examined "one can always find a suggestion of tubular or strandlike arrangement of the cells." Norris[3] states that "the term arrhenoblastoma must, in the present state of our knowledge, refer to a clinico-pathological condition; practically the term must be applied only when both the clinical and the pathological observations jointly justify its use." He says also that "there seems to be no justifiable morphological or genetic reason for deriving this neoplasm from any particular part of the primitive sex gland."

We arrive at these two conclusions: (1) only some of the ovarian tumours showing the supposedly typical structure of a testicular rudiment cause masculinization, (2) tumours which do not show such a structure may yet cause masculinization. It seems that the ill-founded suppositions on which the term 'arrhenoblastoma' was

based are still maintaining certain erroneous ideas as though their truth had been established. In the light of present knowledge it would be rather a gross mistake to suppose that because a tumour produces androgen it therefore should have an architecture like that of the testicle. In the male, androgens are derived from the interstitial glandular cells of the testicle, which have no tubular or strand-like arrangement. This fact alone seems to destroy the foundation of the name arrhenoblastoma and to show that the term as defined in the authoritative literature is bound to cause mental confusion. Vestigial homologues of the vasa efferentia testis are commonly present in ovaries, and an excessive supply of androgen from any source will cause their hypertrophy; a tubular adenomatous structure in the ovary can, therefore, be explained as the consequence and not the cause of masculinization. Some ovarian tumours which induce hirsuties and other masculine phenomena are the colour of corpora lutea and are composed of cells which resemble those of luteal tissue, others might be described from their cytological appearance as thecomas, or as granulosa-cell tumours, and yet others look like tumours derived from adrenal tissue. In fact, by examining sections of an ovarian tumour under the microscope no reliable guidance can be obtained, either from their anatomical arrangement or individual appearance, as to the nature of the hormones, if any, which the cells may have produced.

Two reformations, it is suggested, might be made to render the terminology accord with fact. The first is to follow Ewing[4] and shorten the term arrhenoblastoma to 'arrhenoma' which means 'male tumour' and makes no assumption as to its origin in a testicular vestige; the second is to apply the term to all tumours, whether situated in the ovary, adrenal or elsewhere, which produce excessive amounts of androgen and so bring about virilism. In this sense the name appears etymologically sound and carries an unmistakable meaning free from haziness and fanciful implications.

GRANULOSA-CELL TUMOUR OF THE OVARY.

The objections to this designation are rather like those already adduced against the term arrhenoblastoma. Strictly the term is applied to ovarian tumours the cytology of which recalls the granulosa tissue of ripening follicles and the effects of which are those produced by oestrogen. In practice considerable latitude is shown and oestrogen-producing tumours of the ovary have been described as granulosa-cell tumours though their cytology would not support the title. According to Meyer[1] granulosa-cell tumours do not arise from the granulosa cells of ripening follicles but from undifferentiated cell rests. In some instances, though not in all, the tumours are accompanied by effects like those induced by oestrogen, including hyperplasia of the endometrium and enlargement of the mammae. "Thus," Meyer says, "we have both histologic and hormonal proof that these tumours arise from granulosa cells." Novak and Long[2] agree with Meyer that granulosa-cell tumours arise from cell rests and not from the granulosa of maturing follicles. They say "the granulosa cell is a typical feminine cell, producing the so-called female sex hormone." Against the term as currently used the writer would raise the following objections.

1. Those who have carefully investigated these tumours do not regard them as arising from the granulosa tissue of ripening ovarian follicles. If this opinion is correct the term 'granulosa-cell tumour' conveys a misleading implication.

2. It remains unproved that the granu-

losa cells of normal ovarian follicles form oestrogen; even if they do, they are not the only source. Granulosa tissue is readily destroyed by X-rays without causing other obvious injury to the ovary which will continue to form oestrogen though granulosa cells are absent.

3. Not every tumour which has the appearance of granulosa tissue will cause oestrogenic effects. Some are unaccompanied by evident hormonal disturbance, others cause masculinization.

4. Ovarian tumours composed of cells which do not resemble those of granulosa tissue may yet supply an excess of oestrogen.

From these facts the impediment which the term 'granulosa-cell tumour' offers to clear thought is apparent.

As a remedy the writer suggests that the term 'theeloma' (female tumour) might be used to denote all tumours which cause an excessive supply of oestrogen, whether they originate in the ovary, adrenal or elsewhere.

SUMMARY.

Criticism is made of the terms 'arrhenoblastoma' and 'granulosa-cell tumour' and it is suggested that they might be discarded. It is proposed that the term 'arrhenoma' might be used to denote androgen-producing tumours and 'theeloma' to denote those which produce oestrogen, irrespective of the location of the tumours in the body, or of their histological appearance.

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Spinal Anaesthesia in Cases of Delivery by the Obstetric Forceps

BY

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THE administration of spinal anaesthetics in obstetric conditions when Caesarean section or hysterotomy is necessary is recognized as a useful and beneficial method in suitable cases. The improvement in the solutions and in the technique employed has produced better results so that this method of anaesthesia is coming into more favour than formerly. Rufus Thomas[1] gives a very interesting review of 121 cases of Caesarean section done under spinal anaesthesia and discusses the advantages and disadvantages of the method. Other contributors to the literature on this subject, namely H. K. Ashworth,[2] S. G. Luker,[3] K. G. Lloyd Williams,[4] have commented favourably also on this method of anaesthesia for Caesarean section.

In the Glasgow Royal Maternity and Women's Hospital, spinal anaesthesia has been used for a long time routinely in one unit, and as an alternative method frequently for Caesarean sections and hysterotomies performed by other members of the staff.

The use of spinal anaesthesia in cases of delivery by the forceps has been found to be of benefit under certain conditions.

The cases in which this method would be found to be advantageous are:

1. Deliveries following prolonged labour where there was 2nd stage delay and the patient was becoming exhausted.

2. Cases in which an anaesthetic had been already administered during the course of labour.

3. Cases of cardiac disease especially with an associated chest condition.

4. Cases of albuminuria or other constitutional condition in which a general anaesthetic, especially chloroform, would be contra-indicated.

The cases in which it would not be suitable are:

1. The application of the forceps when the head of the child is high up and the uterus is contracted on an occipito-posterior position of the child. Manipulation to correct this position would be difficult or almost impossible.

2. Any abnormal presentation requiring intrauterine manipulation, e.g. internal or external version (for correction).

3. Any disease of the central nervous system or of the vertebrae or any septic condition at the site of the lumbar puncture.

In administering the anaesthetic the aim is to confine the anaesthesia so far as possible to the level which would affect the perineum and pelvic floor, i.e., a low spinal anaesthesia. It is not necessary to have the skin over the abdominal wall anaesthetized though, as a rule, the anaesthetic affects a narrow strip of skin above the symphysis pubis.

To achieve this result a small dose of a heavy solution is used with the patient in the upright position during the administration. The highest points of the iliac crests are taken and a line drawn between them passes over the tip of the 4th lumbar spine. Injection may be made in the space

above or the space below this point, the easier site being chosen.

In the series of cases reported the earlier ones had planocaine 10 per cent (May and Baker), the specific gravity being 1032 and the later ones had the same preparation in 20 per cent strength with a specific gravity of 1040. The latter solution was found to produce the better result and a dose of 0.5 c.c. to 0.75 c.c. was found to be sufficient.

If blood-pressure is low ephedrine gr. $1\frac{1}{2}$ is injected intramuscularly but as the level of anaesthesia is low any alteration in blood-pressure would be no greater than with a general anaesthetic. The advantages of the method are:

1. The general condition of the patient both during and after operation is much better than with a general anaesthetic.

2. There is much less haemorrhage in the 3rd stage owing to better uterine retraction.

3. The regional nerve block cuts out reflex stimuli from the perineum. This associated with the lessened haemorrhage removes the two main factors which are productive of obstetric shock.

4. The anaesthesia would not produce or aggravate any pulmonary or toxaemic condition.

In the management of the 3rd stage it is advisable not to administer pituitrin before the placenta has been expelled. It was noted that in those patients who had a spinal anaesthetic, pituitrin seemed to cause spasm of the cervix following delivery of the child so that the placenta tended to be retained whereas in those patients to whom pituitrin was not given the 3rd stage was of normal duration and there was no difficulty with the placenta. In the series of 60 cases conducted in this hospital, using spinal anaesthesia for delivery by the forceps, 30 cases were included from Professor Hendry's unit some time ago, and the remaining 30 more recently from Professor Cameron's unit.

The results were as follows:

Parity	1st series	2nd series
Primigravidae	22	26
2-para	6	3
Others	2	1
	30	30

Ages	1st series	2nd series
Under 20 years	3	1
20 to 30 years	17	17
30 to 40 years	8	12
Over 40 years	2	Nil
	30	30

Indications for operative delivery.

First series, mostly 2nd stage delay

Duration of labour under 12 hours	2
12 to 24 hours	8
24 to 48 hours	11
Over 48 hours	9
	30

Second series

Primary uterine inertia	8
2nd stage delay	22
	30

Position of foetus	1st series	2nd series
Occipito-anterior ...	15	8
Occipito-posterior ...	8	6
Transverse arrest ...	7	14
		1 Breech presentation in primigravida.
		1 Twins, 1st vertex; 2nd internal version to breech in primigravida.
	30	30

Operations	1st series	2nd series
Mid forceps	17	20
Low forceps	13	8
Manual breech delivery	0	1
Twins (low forceps internal version manual breech delivery) ...	0	1
	30	30

Infants	1st series	2nd series
Alive	25	25 (2 showed signs of distress)
Stillbirths	3	3
Intrauterine death ...	2	2
	30	30

Maternal condition

1st series	2nd series	
1 death	Good	25
Others satisfactory	Fair	3
	Unsuccessful	2
		30

Among the 60 patients there was only one maternal death, the case being of severe bronchitis with dyspnoea, in which a general anaesthetic was contra-indicated on account of the poor general condition of the patient. The child was stillborn and manual removal of the placenta was necessary. The patient collapsed and died 30 minutes after administration of the anaesthetic.

The cases of stillbirths occurred in infants in which a contributory cause was present either in the general health of the mother or in the duration of labour.

Otherwise the infants were in good condition and did not show any ill-effects from excessive compression by the uterine muscle.

In the second series a manual breech delivery of an infant weighing over 6 pounds was carried out successfully without difficulty, also in a case of twins the second infant was converted by internal version from a vertex to a footling presentation and delivered without difficulty, the first child having been delivered by the forceps. Both patients were primigravidae.

The cases of occipito-posterior and transverse position were converted to anterior positions without undue difficulty before application of the forceps.

Summary and Conclusions.

Sixty cases of operative delivery using spinal anaesthesia are reported and the results in two separate units have been shown in the various tables. Fifty-eight patients were delivered by the forceps, one case of twins by the forceps and internal version followed by manual breech delivery and one case of breech presentation delivered manually.

From the maternal aspect the parity and age group of the patients have been shown, also the indication for operative delivery and the type of operation.

With regard to the infants in the cases quoted the position *in utero* has been noted and the results as regards live and stillbirths.

The conclusions which one may draw from reviewing the cases in both series are that while low spinal anaesthesia would not be advised as a routine method it could be used with advantage in many deliveries by the forceps. Cases which would be unsuitable are those in which intrauterine manipulation to correct the position of the child might be difficult even under general anaesthesia.

The condition of the mother both during and after delivery compares favourably with that under general anaesthesia and in the 3rd stage the loss of blood is much less. In cases in which there might be contra-indications to general anaesthesia the spinal

anaesthetic would overcome the difficulty.

I wish to express my thanks to Professor Samuel J. Cameron and to Professor James Hendry for permission to carry out this method of anaesthesia in their units, also to the Registrar, Dr. Matthew Garrey, for his co-operation.

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Onyalai: A Tropical Condition Characterized by Haemorrhages: Its Gynaecological Aspects

BY

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THE features of the condition known as Onyalai are not to be found recorded in the textbooks of medicine generally used in temperate climates. It is of most interest to the physician but there are certain aspects of Onyalai which are worthy of mention in a journal limited to the study of obstetrics and gynaecology.

In order to give the reader some idea of the chief features of this disease it will be necessary to mention briefly the major medical observations which have been made on Onyalai.

The word Onyalai is used to indicate a haemorrhagic disease, often fatal, which is characterized by the formation of bullae filled with blood in the mucous membrane of the buccal cavity and elsewhere. Most of the cases which have been described were recorded by workers in Central Africa, particularly in Portuguese West Africa. The disease is known to exist outside these territories, but it is very doubtful whether it exists outside Africa. In 1901 Preston Maxwell described a condition which occurred in certain parts of China: this condition appears to have certain points in common with Onyalai. The cause of Onyalai is not known. The subjects of the disease are young adults and men seem to be affected more often than women. Onyalai has not been described in Europeans resident in tropical and sub-tropical countries.

CLINICAL FEATURES.

Onyalai develops suddenly. The initial symptoms are pains in the limbs and headache: the temperature rises in the majority of cases. The worst cases are those which have a subnormal temperature.

The tongue becomes painful and colicky abdominal pain is often present. Bleeding occurs rapidly, that is, a few hours after the initial symptoms. Bleeding is most common from the nose and mouth; conjunctival haemorrhages are also frequently seen. Haematuria occurs in a large proportion of cases. The parotids tend to be swollen and tender.

Examination of the patient reveals the presence of haemorrhagic bullae which vary from the size of a pin's head to about a centimetre in diameter. Such bullae may be seen on the inside of the lips and cheeks, on the tongue, palate and fauces. The bullae also occur in the respiratory tract, in the intestinal and urinary tracts and extensive lesions are generally present in the skin. The bullae, which contain dark blood, break readily and this appears to be the cause of loss of blood from the organs mentioned above. The deeper tissues are also affected by Onyalai. Haemorrhagic lesions arise in the lungs, the brain, the liver, and haemorrhagic effusion appears in the serous cavities. Haemorrhages may occur in the spleen: it is unusual for this organ to be enlarged in Onyalai.

The course of the disease is very variable. Some patients have profuse haemorrhage from the nose and mouth, respiratory, intestinal or urinary tracts and death is rapid. In other patients the lesions are not extensive and there is only a slow ooze of blood: these patients generally recover.

Examination of the blood in cases of Onyalai reveals a sudden fall in the red cells: the resulting anaemia may be very severe. The blood platelets drop suddenly at the beginning, occasionally falling to less than 1,000 per c.mm. There is a leucopenia and the reticulocyte count is low. The coagulation time is normal, but there is marked prolongation of the bleeding time.

If recovery takes place the platelet count rises rapidly and the bleeding ceases. The count does not reach normal until a few weeks have expired. When bleeding ceases the red blood cells start to rise in numbers.

Onyalai is therefore considered by those who have studied this disease to belong to the group of purpuras: it is regarded in the light of an acute thrombocytopenia.

In the present state of knowledge the treatment resolves itself into the administration of donor's blood. In the severe cases blood transfusion may be necessary, but in the milder cases intra-muscular injection of 20 c.c. of citrated blood given once or twice a day is usually sufficient. It may be necessary to continue the injections for 5 or more days. Calcium is often given, but it is not clear whether it is of much help.

The Gynaecological Aspect.

From the gynaecological point of view it is of interest to note that haemorrhage also takes place from the genital tract. In the few cases which the writer has observed, vaginal bullae were discovered on one occasion and these appeared to be the source of the blood-stained discharge. In another 2 cases blood was seen issuing from the external os. Not every case of Onyalai in the female bleeds from the genital organs; this is true of both the severe and the mild types. Insufficient material has been seen to determine whether the uterine bleeding in Onyalai occurs in the early or late phases of the menstrual cycle. In one case in which the disease occurred in a multipara it was possible to obtain a small piece of endometrium for biopsy without unduly disturbing the patient. When examined microscopically the endometrial picture was typical of the first half of the cycle. There was diffuse haemorrhage in the whole of the small piece of tissue taken for section and it was impossible to see anything other than the normal arrangement of glands and stroma and interstitial haemorrhage.

Another patient (No. 464, 1942) was sent into hospital from a country district. This patient was a young nullipara. On examination haemorrhagic bullae were found on the lips, on the tongue, the inside of the cheeks and in the skin. Blood was not present in the urine. The patient was losing profusely *per vaginam*, the loss necessitating the use of 16 pads a day. The temperature oscillated about 101° F. Examination of the blood showed that the platelets were reduced to 8,760 per c.mm. Vaginal examination did not reveal any abnormality of the genital tract. There was no suspicion of pregnancy. The patient stated that she believed her next menses to be due about the time when she started to lose blood from the nose and mouth. Natives give notoriously bad histories and too much reliance cannot be placed on this. This patient was treated with intra-muscular injections of donor's blood and Coagulen Ciba. Recovery took place in 10 days, the loss of blood *per vaginam* ceasing about the 9th day. No history of excessive or irregular menses could be obtained. The stools and urine were examined and found to be free from parasites.

Onyalai is not a common disease and it is decidedly rare for it to be seen during pregnancy. One case of Onyalai in a pregnant multipara has been seen by the writer. In this case the symptoms devel-

oped about the 36th week of pregnancy. On admission the symptoms were found to be severe: treatment was carried out in the usual way. The foetus was alive and the pregnancy was normal so far as could be determined. On the 4th day of treatment the husband insisted on taking his wife to his kraal on the grounds that the time of delivery was near. The patient returned to hospital about 4 days later with severe bleeding from the nose and mouth and haemorrhages in the conjunctivae and skin: there was no vaginal bleeding. Examination revealed the foetus to be alive and the uterus felt normal to the touch. The patient was given a blood transfusion and the nose was plugged. Two days later labour commenced and progressed normally, a live baby being delivered. No excessive loss of blood took place during the 3rd stage of labour and the baby appeared to be normal in all respects. After delivery the mother's condition improved rapidly, bleeding from the nose and mouth stopping on the 2nd day of the puerperium. No information could be obtained about subsequent events as it is impossible to "follow-up" native cases.

Summary.

Onyalai is a haemorrhagic disease of the nature of an acute thrombocytopenia. One of the symptoms may be bleeding from the genital tract; the blood may come from vaginal lesions or from the endometrium. In the solitary case in which Onyalai has been observed in late pregnancy it did not interfere with the pregnancy or the labour.

Although the disease has so far attracted attention only in Central Africa it is possible that it occurs in other places with similar climatic conditions, such as some parts of Central and Southern America. The description of these cases may induce others who have made similar observations in other countries to record their findings.

Fibroma of the Ovary with Ascites and Hydro-Thorax

BY

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THE presence of a pelvic tumour associated with ascites and pleural effusion is immediately suggestive of inoperable growth. That such a syndrome may exist with a benign and easily operable neoplasm of an ovary, it is important to recognize. Only 17 such cases have been described in the literature making 18 with that to be reported. This would suggest great rarity. It is more likely, however, that many others exist. Some of these may well have been denied operation when the existence of the syndrome was not recognized.

The first case was reported by Cullingworth[1] in 1879. There is no further reference till 1923 when Hoon[4] of the Mayo Clinic reported 2 cases. Meigs[8, 9, 10] in 1934 referred to 3 cases, reported a 4th in 1937 and subsequently collected 17 from the literature and from personal communications.

Rhoads and Terrell[11] first used the term "Meigs' Syndrome" when reporting a case in 1937.

SYMPTOMS.

In the majority of cases the main symptoms were referable to the chest. The chief complaints were of shortness of breath, fatigue, cough and pain in the chest.

PHYSICAL EXAMINATION.

There was usually considerable emaciation and evidence of loss of weight and dehydration. There may be some relation between this dehydration and the rapid reaccumulation of fluid observed after tapping. The fluid in the chest was most often on the right side. In 3 of 18 cases it was on the left. One side only would appear to be affected. There was no relation between the side of accumulation of the fluid and the ovary involved.

The tumours are not massive ones, so that in the presence of much ascitic fluid it may be difficult on physical examination to determine that one is dealing with a smooth rounded mobile ovarian mass.

CLINICAL, PATHOLOGICAL FINDINGS.

The specific gravity of the fluid varies between 1.013 and 1.018. Evidence of tuberculosis has not been found on guinea pig inoculation.

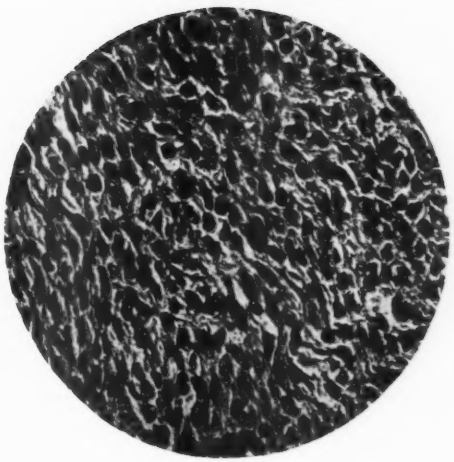
Only in the case here reported was there a high eosinophil content in the pleural fluid.

OPERATION.

In most cases the chest was tapped before operation. Simple removal of the tumour is all that is called for. All the cases operated on have recovered and the hydrothorax and ascites rapidly and permanently disappeared.

PATHOLOGY.

The tumour in each case was a typical fibrous connective tissue tumour with many collagen fibres.



A.G.

Case Report.

W. C., a married woman, aged 30, was admitted to hospital complaining of cough, shortness of breath and loss of weight over a period of 2 months. She also had pains across the abdomen and attacks of diarrhoea. Her bowels had previously been regular. Menstruation had always been regular with a normal loss lasting 5 or 6 days every 28 days, but she had missed her last period which should have appeared a fortnight before admission. There had not been any pregnancies.

On examination the patient was seen to be very emaciated, with sunken eyes, hollowing of the cheeks, lax skin and pasty complexion. The pupils reacted to light and accommodation. There was no abnormality in the neck, other than that the trachea was slightly away from the mid-line to the left. There was lessened movement and vocal fremitus was decreased on the right side of the chest. The percussion note was dull anteriorly and posteriorly and breath sounds were absent on the right side. The pulse was regular. The apex beat was situated in the 5th intercostal space a half inch lateral to the nipple line. The heart sounds were normal, and the blood-pressure was 110 systolic, 75 diastolic, mm. of mercury.

The abdomen was very distended with eversion of the umbilicus and dullness in the flanks which changed on movement. A mass could be felt arising from the pelvis on the right side for a height of 5 cm. above the inguinal ligament.

On vaginal examination there was a hard mass in the posterior and right fornices but the body of the uterus could not be made out separately from the mass, nor could the mass be moved on bi-manual examination.

On rectal examination a mass could be felt high up on the right side.

An X-Ray of the chest showed a massive effusion on the right side. After aspiration of 1,500 c.c. of fluid and replacement with air some re-expansion of the lung occurred. X-Ray of the re-expanded lung did not show any evidence of secondary deposits. The pleural fluid was opalescent and contained an excess of cells of which a few were endothelial, the remainder leucocytes. Eighty-two per cent of these white cells were eosinophils, 14 per cent leucocytes and 4 per cent mast cells. The fluid was sterile on culture and tubercle bacilli could not be detected in film or culture.

Examination of the blood showed a red cell count of 4,060,000, haemoglobin of 60 per cent (Haldane) and colour index of 0.7. Differential white count showed a neutrophil leucocytosis of 89 per cent but there was no increase in the total white cell count.

Operation was performed under cyclopropane anaesthesia through a mid-line suprapubic incision. A considerable volume of clear yellowish ascitic fluid was sucked away. The small bowel was collapsed and injected and some of the loops which had been lying in the pelvis were matted together with filmy adhesions of partly organized coagulated exudate.

There was a smooth, shiny, whitish mass 25 cm. × 20 cm. × 15 cm. attached by a short pedicle to the posterior aspect of the right broad ligament. On the surface of the mass the flattened right ovary was seated.

The pedicle was clamped, divided and ligatured. So far as could be determined, by passing a hand above and behind the liver, there was no hole in the diaphragm.

Convalescence was uneventful. There was no further re-accumulation of ascitic fluid and 3 weeks after operation the fluid had disappeared from the right side of the chest.

The section of the tumour of which a microphotograph is reproduced, was examined by Dr. J. R. Gilmour who reported "A spindle celled fibroblastic tumour. A normal ovary was attached to the growth suggesting an origin in the mesovarium. The growth was very cellular but mitoses were rare, probably a fibroma."

Six months after the operation the patient was enjoying normal health and X-ray of the chest showed normal lung fields.

Comments.

There are two interesting features of this case which do not appear in any of the 17 cases previously described in the literature. Firstly, there is the very high eosinophil content of the pleural fluid and secondly, the suggestion that the fibroma had arisen in the mesovarium.

A reasonable suggestion as to the origin of the ascitic or pleural fluid cannot be offered. Even if one were to accept the

explanation of an " irritative " effect of the fibroma on the peritoneum this still leaves the pleural fluid to be accounted for. The suggestion that a communication between peritoneal and pleural cavities through the diaphragm must exist, or that venous drainage of the pleural cavity by the azgos veins is interfered with, is entirely unsupported.

SUMMARY.

1. The clinical features of the syndrome of fibroma of the ovary with ascites and hydro-thorax (Meigs' syndrome) are summarized.

2. Attention is drawn to the possibility of the syndrome being regarded as due to malignant metastases with consequent denial of operation.

3. A case of the syndrome is described.

ACKNOWLEDGEMENT.

Thanks are due to Dr. D. H. Irwin, Medical Superintendent, Essex County Hospital, Wanstead, for permission to publish the case.

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Soap as a Foreign Body in the Bladder

BY

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AND

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ACCORDING to Badenoch and Campbell[1] about a dozen papers are published every year on the occurrence of foreign bodies in the bladder. Tcherlok,[2] in an extensive review, found that foreign bodies were not usually introduced in attempts to procure abortion, but most of the cases among women reported from this country have been of this nature (Christie,[3] Farncombe[4] and Charles[5]). Foreign bodies in the bladder are almost always hard and coated with a phosphatic concretion, and their presence leads to haematuria, infection and severe pain on micturition. Numerous papers have appeared, mainly in the French journals, on the effect of intrauterine injection of soap solutions (Yovanovitch,[6] and Gross[7]), but we have not found any record of the introduction of soap into the female bladder. A woman has recently been seen in this hospital whose urine contained fatty acids and dissolved soap which, according to clinical observation, were present in the urine during its passage down the urethra.

CASE REPORT.

An unmarried woman, aged 20 years, complained of pains in the back of 2 weeks' duration, with dysuria and frequency of micturition. Her recent history was of "nephritis" in 1940 with haematuria and scalding pains on micturition, but no oedema. In 1941 she was in hospital with pain in the right iliac fossa, but there was no diagnosis. Early in 1942 she had pleurisy on the right side.

Clinical examination. The patient looked well but appeared to be in pain. There was considerable tenderness on deep palpation of the kidney region on both sides, with some tenderness in the right iliac fossa. Rectal examination revealed tenderness apparently in the region of the junction of the right Fallopian tube and the uterus. The menstrual history was of a 29-day cycle with 3-day periods which had recently been scanty and very painful.

Pathological examination. The blood urea and the chlorides, alkali reserve and proteins of the plasma were normal, and so was the blood count. The urine, *p*H 6.5, contained albumin and acetone bodies and a film of the deposit showed numerous Gram-negative bacilli which on culture produced a heavy growth of B. coli. The patient, who was not a virgin, had no urethral discharge, nor were there any signs of venereal disease.

Treatment. The B. coli infection was successfully treated by a week's course of "Albucid" (7.5 grm. per day) but the tenderness in the region of the Fallopian tubes continued, accompanied by a temperature which varied between 97°F. in the morning and 103°F. in the evening. Since this condition did not respond to sulphapyridine therapy a gynaecological examination under a general anaesthetic was performed, but did not reveal any abnormality.

Fatty contaminant of urine. An oily substance which solidified on cooling had been noticed on the surface of the urine ever since admission, and it was found that this substance was soluble in ether and chloroform. When crystallized from the latter solvent it formed whorls of needles melting at 36°C. to 37°C. which gave an acid reaction when pressed on wet litmus paper and liberated CO_2 from sodium bicarbonate solution. The crystals had a strong smell of scented soap and it was

suspected that they were a mixture of fatty acids. They were therefore dissolved in caustic soda, acidified, and the solution extracted with ether, evaporation of which yielded pale yellow needles; melting point 31.5°C. to 34.2°C.; setting point in melting point tube 28.0°C. to 26.9°C.; equivalent weight 263. Acidification of the aqueous residue after ether extraction of the urine yielded a similar mixture of fatty acids, equivalent weight 268.

Apparently the urine had contained floating droplets of free fatty acids, together with dissolved soap. Confirmation of this was obtained by the isolation of the fatty acids from a specimen of the patient's toilet soap. The odour and crystalline form were exactly the same as those of the substances isolated from the urine: melting point 32.5°C. to 39.5°C; setting point 32.5°C. to 29.2°C.; equivalent weight 266. It is not surprising that the melting and setting points of the acids isolated from the urine should be somewhat lower than those of the fatty acids derived from the soap since the former were yellow in colour and would contain fat-soluble substances from the urine. The close correspondence between the equivalent weight of the fatty acids from the soap with that of the acids from the urine leaves little doubt that they were identical. The toilet soap did not contain any free fatty acid, but after incubation at 38°C. for 48 hours with urine (*p*H 5.5) the semi-solid floating matter, when extracted with ether, yielded fatty acids of equivalent weight 243.

When the patient was deprived of her soap, floating matter disappeared from the urine and a 24-hour urine specimen taken 2 days after the one which had yielded the results just described contained neither free fatty acids nor soap. The positive reaction for acetone bodies continued for about a week, but as the food intake was abnormally low the acetonuria cannot definitely be correlated with the presence of soap in the bladder. The swinging temperature was eventually found to be associated with a hot-water bottle under the patient's pillow. Examination by a psychiatrist gave no further information and the patient was discharged with a normal urine and temperature and without any abdominal pain.

Conclusion.

The chemical evidence presented leaves little doubt that the floating matter found in the urine consisted of fatty acids derived from the patient's toilet soap. The hypothesis that this material came from the bladder rests largely upon the observation that the surface of the freshly-passed urine was covered with oily drops of colourless liquid resembling liquid paraffin, which solidified as the urine cooled. Soap incubated for 48 hours with urine swelled up considerably and was partially converted to fatty acids, but did not completely liquefy, so that the oily drops could hardly have been produced by dropping the soap into the urine after it had been voided.

Summary.

A case is described in which a patient is believed to have introduced toilet soap into her bladder. The soap was voided partly in the form of fatty acids which floated on the surface of the freshly-passed urine in the form of colourless oily droplets.

Acknowledgments.

Our thanks are due to Dr. J. Mills for assistance in revision of the manuscript, and to Dr. L. M. Jennings for permission to publish the clinical records.

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The Advantages and Disadvantages of Trial Labour

BY

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Honorary Assistant Obstetrician, Princess Mary Maternity Hospital, Newcastle upon Tyne; Consulting and Visiting Gynaecologist, Richard Murray Hospital, Blackhill, Co. Durham.

TRIAL labour is primarily intended for cases of moderate disproportion in primigravidae in which it is considered that, if the patient co-operates satisfactorily, the uterine action is efficient and the normal process of adaption of the foetus to the birth canal during labour is not disordered, delivery *per vaginam* will be practicable without undue risk of damage to mother or child. Its chief object is to minimize avoidable interference with normal course of labour.

This study is based upon a series of 127 consecutive cases of trial labour conducted in the Princess Mary Maternity Hospital during the past 7 years. About 20 to 25 per cent of the patients were under the care of Mr. Frank Stabler and the remainder were my own. In order to form a basis for comparison, patients in whom the true conjugate was estimated to exceed $3\frac{3}{4}$ inches (whatever the size of the child), in which the true conjugate estimate was not recorded, in which the baby weighed less than 6 pounds, and in which a previous pregnancy had been carried beyond the 7th month were excluded. All patients had been examined during the late antenatal period or, in the few emergency cases, at the onset of labour, by the obstetrician in charge of the case.

The results observed are tabulated, for comparative purposes, with figures obtained from a series of 100 primigravidae without any evidence of disproportion:

	Cases	Stillbirths	Neonatal deaths	Maternal deaths	Notifiable pyrexia
Trial labour ...	127	3.1 per cent (4 cases)	2.4 per cent (3 cases)	0.8 per cent (1 case)	2.4 per cent (3 cases)
Normal labour...	100	1 per cent	1 per cent	0 per cent	1 per cent

		Normal delivery	Instrumental delivery	Caesarean section	Duration of labour
Trial labour ...	...	53.5 per cent (68 cases)	31.5 per cent (40 cases)	15.0 per cent (19 cases)	31 hours
Normal labour	...	87 per cent	13 per cent	0 per cent	21 hours

From time to time objections to the procedure, of which the following are the most plausible, have been raised.

1. *The use of trial labour is a confession of failure on the part of an obstetrician who is unable to assess the degree of disproportion in his patients and therefore evades the*

issue by trying his luck with a course which will not commit him to a definite decision.

This objection applies to the abuse and not the use of trial labour. Cases for trial must be selected with great care, and a patient must not be permitted to embark upon such a labour without a thorough investigation and careful assessment of the case. As, in spite of the most detailed examination, it is impossible to foretell with accuracy the fortitude of the patient, the efficiency of the uterine action or the precise mechanism which will operate during labour, the most rational way to find out how labour will progress in a border-line case of disproportion is to give a fair test. The selection of cases for trial and the management of labour have been discussed elsewhere.[1, 2]

2. *The teaching of methods of trial labour in medical schools may be misconstrued by some practitioners who, trusting to the trial to solve their problems, may neglect to examine their patients thoroughly before labour commences. After a short and inadequate trial labour such practitioners tend to lose confidence in their patients, and in themselves, and to perform that which is, only too often, an unnecessary Caesarean section.*

The choice of patients for trial labour and their management during the course of labour is outside the province of the general practitioner. Every patient should be under the immediate supervision of an experienced obstetrician who should assume sole responsibility for any decisions which may be necessary regarding the conduct or termination of labour. The selection of cases and choice of technique for Caesarean section is the duty of the obstetrician and not of a general practitioner or a general surgeon. The 15 per cent cases which require Caesarean section after trial labour might possibly have been delivered *per vaginam* had induction of premature labour been performed in 100 per cent cases at the cost of the lives of babies.

3. *The unfavourable psychological effect upon the mother of the death of her first baby after a long and distressing trial labour may have far-reaching consequences, and in these cases induction is valuable because, if the pregnancy is allowed to proceed to term, it is a matter of chance if the baby can be delivered alive.*

After excluding patients delivered by Caesarean section and those in whom the time of onset of labour was not recorded, the average duration of the 1st and 2nd stages of labour in my trial cases was 31 hours compared with 21 hours in my normal cases. Peel[3] records the duration of labour after bougie induction in 90 cases as 32 hours 49 minutes.

In my trial labour series the stillbirth-rate was 3.1 per cent and the ten-day neonatal mortality-rate 2.4 per cent. Townend,[4] a supporter of premature induction, puts the combined foetal and neonatal mortality following this treatment for minor and moderate disproportion at 10 per cent, Bainster[5] at 12.6 per cent, and Eden (quoted by Holland) at 14 per cent. Holland[6] epitomizes the case against induction in the following words: "The wholehearted exponents of induction . . . spread their net wide. They are willing to induce many cases unnecessarily, in order to catch one in which the operation has been done at the proper time and has saved a Caesarean section or a craniotomy or a too difficult forceps extraction at term."

4. *Trial labour in domiciliary midwifery is not only worrying to the patient, distressing to her relatives and harassing to the nurse but irksome and exhausting to the practitioner in charge of the case.*

Trial labour should always be conducted in a hospital where facilities will be available for proper nursing attention, for effective antiseptic precautions and for any

operative procedures which may prove necessary during the course of labour. It is wholly unsuited to domiciliary practice. Peel replies to the last point in the words: "If the doctor argues that he cannot afford the time to bear with the uncertainty these difficult cases often occasion, he is not a suitable person to deal with them."

5. *Delivery by the forceps is frequently necessary for the termination of trial labour.*

In my opinion the forceps-rate (in my series 31.5 per cent) is not unduly high in this class of case, and I think it is capable of considerable reduction by more frequent use of episiotomy during the 2nd stage of labour.[7]

6. *The maternal morbidity- and mortality-rates in patients subjected to trial labour are high.*

In my series of cases the notifiable pyrexia-rate was 2.4 per cent and the maternal mortality-rate 0.8 per cent, figures which, in my opinion, are not excessive for cases of disproportion.

7. *Since the maternal mortality is known to rise the later in labour Caesarean section is performed the tendency will be to do it early, with the result that, instead of unnecessary inductions, there will be unnecessary Caesarean sections.*

Indications for Caesarean section during trial labour are lack of advance of the foetal head with the cervix more than half dilated in spite of satisfactory contractions an hour or more after rupture of the membranes, excessive moulding and caput formation without progress, and early evidences of obstructed labour. Signs of maternal or foetal distress becoming evident while the greatest diameter of the foetal head remains above the pelvic brim sometimes hasten the decision, as may also increasing oedema of the cervix, disordered uterine action and lack of intelligent co-operation on the part of the patient. If these indications are taken as a guide, few unnecessary Caesarean sections will be performed, provided that the cases for trial have been properly selected.

8. *The educational value of labour following premature induction is as great as of trial labour at term and observations made during premature labour are equally helpful in arriving at a decision regarding the management of subsequent pregnancies.*

This sweeping statement bears no relation to the facts or even to reason. No true 'test' is involved in the average premature labour and the mechanism in such cases is atypical. It would be almost as rational to argue that, as a golf ball can roll into a standard hole, a biased bowl can do likewise.

I therefore conclude that trial labour is a justifiable and helpful procedure in cases of moderate cephalo-pelvic disproportion in the primigravida and that it is not attended by undue maternal or foetal hazard.

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A Case of Anuria Following Manual Removal of Placenta and Blood Transfusion with Subsequent Development of Irregular Heart Action Cured by Potassium Administration

BY

REGINALD A. E. MAGEE, M.B., B.Ch., B.A.O. (Belfast).

Senior Tutor in Obstetrics, Queen's University and Royal Maternity Hospital, Belfast.

Mrs. D., aged 35, 5-para, whose previous obstetric history showed:

1935. Delivery by the forceps at term, postpartum haemorrhage, blood transfusion; child alive and well.

1936. Five months miscarriage.

1938. Seven months birth. Retained placenta and manual removal; postpartum haemorrhage; child died, 1 day.

1940. Six months stillbirth; retained placenta and manual removal; postpartum haemorrhage.

During the antenatal period of the present pregnancy the patient was treated for pyelitis 4 weeks before delivery, being given 16 grm. M. & B. 693 over a period of 2 weeks.

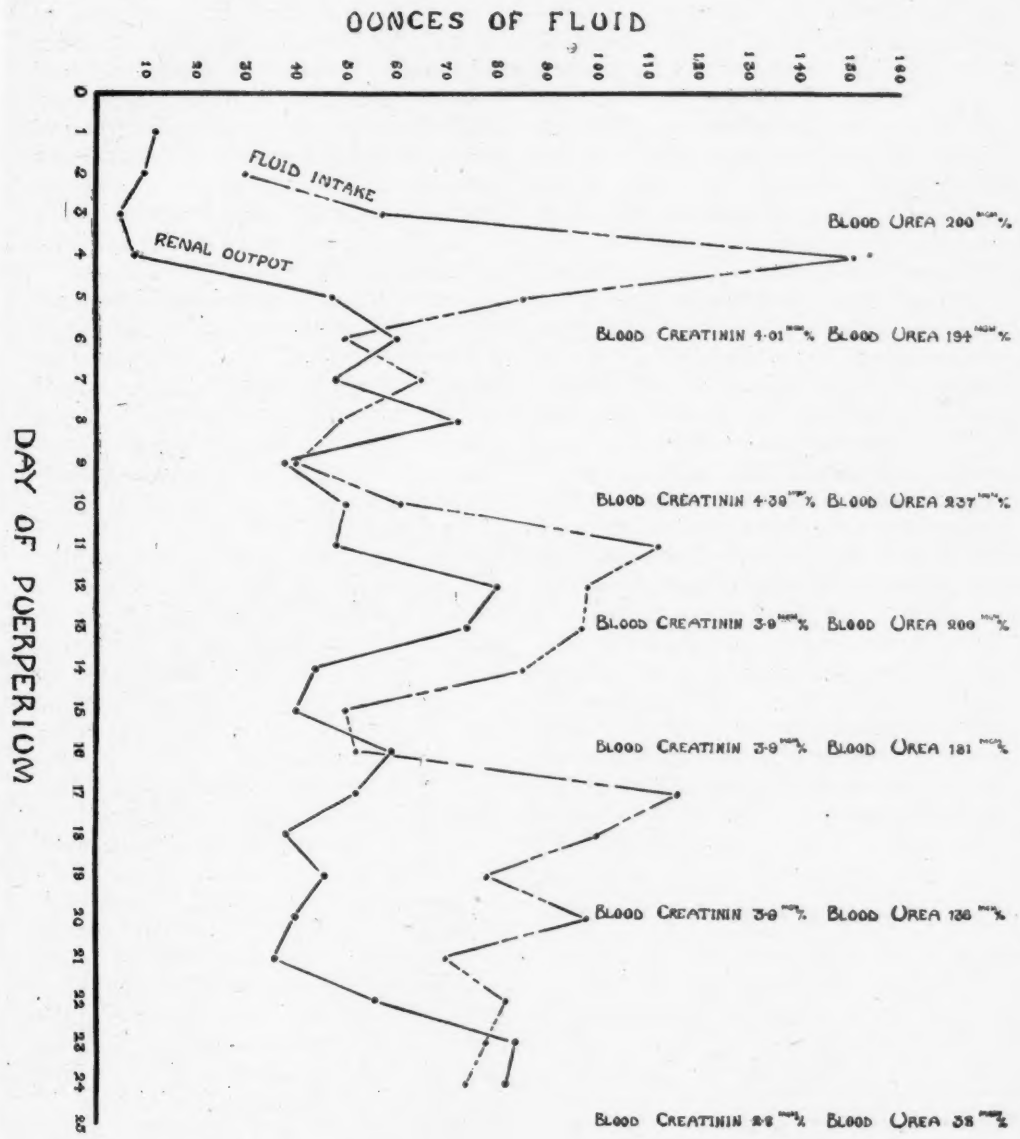
She was admitted to hospital on August 2nd, 1942, having been delivered of a stillborn child at her own home 8½ hours previously; she had a severe postpartum haemorrhage with retained placenta. The placenta had been removed manually by her own doctor who, however, was not satisfied that he had completely removed it. She was given 50 c.c. 50 per cent glucose intravenously, morphia gr. ¼ and ergometrine and transferred to hospital.

On admission 6 hours after manual removal her condition was poor, pulse 140, weak; blood-pressure 62/40; temperature 98.2, anaemic, faint icterus with only slight bleeding. Morphia gr. ¼, intravenous glucose-saline drip and blood transfusion were given. Group O, cross typed. During the blood transfusion the patient was anaesthetized with ether and a manual exploration of uterus performed 2 hours 20 minutes after admission. Placental shreds were removed with the finger and a hot intrauterine douch given. Bleeding was not excessive. Pitocin and ergotrate were given and the blood transfusion completed. The pulse-rate after operation was 120; blood-pressure 86/48: later the pulse-rate rose to 140 and the blood-pressure fell to 60/35. Morphia gr. ¼ was repeated and oxygen and coramine were administered. Six hours later the pulse-rate was 122, and the blood-pressure 80/48, the improvement being maintained. The following day she developed almost complete anuria which persisted for 4 days.

Day of puerperium	Urine passed in 24 hours	Fluid intake
1st	12 ounces	? ounces
2nd	10 ,,	20–30 ,,
3rd	5 ,,	57 ,,
4th	8 ,,	151 ,,

The blood urea rose to 200 mg. per cent on the 3rd day of the puerperium and 206 mg. on the 4th day.

On the 2nd day of the puerperium she was given 100 c.c. 50 per cent glucose solution intravenously. On the 3rd day


OUNCES OF FLUID
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25
DAY OF PUERPERIUM
FLUID INTAKE
RENAL OUTPUT
BLOOD UREA 200 mgm%
BLOOD CREATININ 4·01 mgm% BLOOD UREA 194 mgm%
BLOOD CREATININ 4·38 mgm% BLOOD UREA 237 mgm%
BLOOD CREATININ 3·9 mgm% BLOOD UREA 200 mgm%
BLOOD CREATININ 3·9 mgm% BLOOD UREA 181 mgm%
BLOOD CREATININ 3·9 mgm% BLOOD UREA 126 mgm%
BLOOD CREATININ 2·6 mgm% BLOOD UREA 38 mgm%

Fluid intake and renal output chart showing levels of blood urea and blood creatinin.

continuous administration by the intravenous drip method of normal glucose-saline was commenced with 100 c.c. of 50 per cent glucose per 560 c.c. 5 per cent glucose-saline. A separate transfusion of 200 c.c. 4.3 per cent sodium sulphate was given. The drip was discontinued on the 6th day.

She was also given alkali orally in the form of sodium citrate and Tab. Alk. Effervesc. Co. (B.W.) but she was vomiting intermittently and it is doubtful if any of these drugs were retained. Antiphlogistine was applied to the loins.

On the 5th day urinary secretion recovered, the amount passed being 47 ounces in 24 hours. See graph of fluid intake and renal output, page 449. During this day 0.5 c.c. Salyrgan was given and repeated the following day. For the next 5 days although the urinary output averaged 50 ounces per day its urea content never rose above 1.10 gr. per cent, the blood urea gradually rising to 237 mg. per cent and the blood creatinin to 4.39 mg. per cent. Some degree of pulmonary congestion had appeared but responded to atropine and digitalis. On the 9th day vomiting made it necessary to resume intravenous glucose-saline to maintain fluid intake.

Day	Intake (ounces)	Output (ounces)	Vomit (ounces)
5th	85	47	5
6th	50	60	–
7th	65	48	–
8th	49	72	18
9th	40	38	25
10th	61	50	10

Her general condition had now deteriorated. The stomach was washed out and sodium bicarbonate solution left in to allay vomiting.

On the 12th day her haemoglobin was 35 per cent; red-blood corpuscles 1,280,000 and white-blood corpuscles 11,000, and on the 14th day the intravenous glucose-saline drip was discontinued because of local phlebitis; intravenous therapy having extended over 9 days. Two days later a rectal glucose drip was commenced. The urinary urea was still low, 0.75 gr. per cent in concentrated specimen. Blood urea had fallen to 181 mg. per cent and blood creatinin to 3.90 mg. per cent.

On the 18th day her condition appeared hopeless, she was much weaker, complaining of headache and thirst. Her pulse-rate fell to 36, the heart action being very rapid and irregular. It now appeared that the myocardium was failing—the pulse-rate varying between 36 and 60. This failed to respond to cardiac stimulants.

Bearing in mind the physiology of heart muscle contraction it was considered that this irregularity might be due to an imbalance between Ca,K. and Na ions in the plasma. Consequently on the 20th day 500 c.c. Ringer's solution was injected by the retromammary route and several hours later the pulse-rate was 72, the heart-rate and rythm normal.

On the 21st evening the irregularity of heart had returned and calcium was then given in the form of Calcium Sandoz intramuscularly in 5 to 10 c.c. doses. No improvement in heart irregularity occurred.

On the 23rd day a further 300 c.c. of Ringer's solution was injected by retromammary route and 4 hours later the pulse- and heart-rate and rhythm were quite normal. As large amounts of Na had been given in intravenous glucose-saline drip potassium was now administered in the form of potassium citrate and potassium bicarbonate grains 40 2-hourly: the following day the heart-rate and rhythm were regular and normal and remained so from that time onward.

After 2 days of the potassium therapy the

serum calcium was 11.0 mg. per cent; serum potassium 21.0 mg. per cent.

On the 25th day her condition was very much improved. Blood urea 38 mg. per cent; blood creatinin 2.8 mg. per cent and by September 7th, 1942—the 36th day—the blood urea had fallen to 28 mg. per cent and the blood creatinin to 2.2 mg. per cent. See graph, page 449.

The following investigations were then carried out to determine renal function:

1. Catheter specimen of urine—persistent pus and B coli.
2. Retrograde pyelogram: No definite evidence of pyonephrosis although the pelvis, right kidney, and ureter were somewhat dilated.
3. Uroselectan pyelogram: filling indefinite.
4. Ureteric catheter specimens of urine:
   Right: erythrocytes, urea: 640 mg. per cent.
   Left: erythrocytes, pus cells, epithelium, urea: 540 mg. per cent.
5. Standard urea clearance: 15 per cent.
6. Blood urea: 25 mg. per cent.
7. Blood-pressure: never rose above 110/64.
8. Urea concentration test:

Specimen		Specific gravity
September 3rd, 1942 (32nd day)		
1	Urea 1.42 grm. per cent	1014
2	,, 0.51 ,, ,, ,,	1007
3	,, 0.67 ,, ,, ,,	1009
September 7th, 1942.		
1	Urea 1.6 ,, ,, ,,	1012
2	,, 0.25 ,, ,, ,,	1006
3	,, 0.35 ,, ,, ,,	1010
September 26th, 1942.		
1	Urea 1.85 ,, ,, ,,	1020
2	,, 0.6 ,, ,, ,,	1005
3	,, 0.72 ,, ,, ,,	1005

On discharge, September 26th, 1942, following intensive iron and liver therapy her haemoglobin was 63 per cent red-blood corpuscles 4,210,000.

Discussion.

There was no reason to suppose that the blood transfusion had been incompatible even though a previous transfusion had been given. The patient was of group A and Rh positive and the donor group O, the blood was typed directly and did not show any agglutination. It is interesting to note that the husband was also Rh positive.

When the renal output did return to normal on the 5th day its urea content was very small and the blood urea continued to rise. It was not until the 20th day that the blood urea and creatinin fell appreciably.

It is possible that the large quantity of NaCl given in the intravenous drip may have altered the mineral ion balance of the plasma displacing K possibly by being excreted with Na, thus leaving Ca in ascendency—the heart contraction thus being affected.

In the absence of repeated serum potassium estimations there can be no proof that this conclusion is correct, but the behaviour of the heart after the administration of potassium is evidence in support of it.

That Na does displace K has been shown by Van Slyke[1] who states "when diuresis is provoked by ingestion of either K or Na salts there appears normally to be an acceleration of the hourly output, not only of the base which has been taken but also of the other—an acceleration of K output occurs after a dose of NaCl."

In this case the action of Ringer's solution was dramatic, that of potassium salts curative.

It is interesting to note in this connexion that D. R. Hooker[2] demonstrated the efficacy of potassium chloride in over-

coming ventricular fibrilation induced in dogs by electric shock.

Summary.

1. A case of anuria for 4 days following manual removal of placenta in which a blood transfusion had been given is described. The blood creatinin rose to 4.39 mgm. per cent.

2. Recovery of renal function after prolonged intravenous therapy.

3. Myocardial failure possibly due to lack of potassium in serum as a result of prolonged intravenous NaCl.

4. Temporary recovery of heart irregularity following retromammary Ringer's solution.

Permanent recovery of heart irregularity following potassium therapy.

Acknowledgment.

I wish to thank Professor C. G. Lowry for his kindness in permitting me to publish this case.

References.

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2. Hooker, D. R. "Recovery of the heart in electric shock," *Amer. Journ. Physiol.*, 1929, xci, 305.

ROYAL COLLEGE OF OBSTETRICIANS AND GYNAECOLOGISTS

THE Quarterly Meeting of the Council was held on Saturday, October 23rd, 1943, in the College House, with the President, Sir William Fletcher Shaw, in the Chair.

The following was elected to the Membership of the College:—

Professor John Joseph Kearney, Cork.

The following were admitted to the Membership:—

John Kingston Baker.
Johan Kruger.
Katharine Isabel Liebert.

It was announced that the donor of 1,000 guineas to commemorate the Presidency of Sir William Fletcher Shaw wished the interest to endow a lecture every second year to be given alternately (1) by a member of the College on the subject of infertility, and (2) by a gynaecologist from the U.S.A. as an expression of the friendship existing between the two nations.

The following have satisfied the examiners and have been awarded the DIPLOMA of the College:—

Agnes Urquhart Campbell.
William Harwood Carlisle.
James Thomas Carroll.
James McDiarmid Corston.
Margaret Christian Sandilands Crocket.
Rudolf Walter Danziger.
Grace Trepass Dawson.
Mary Elizabeth Egerton
Charlotte Leopoldine Hess.
Peter Cyril Lewis.
Eileen Cathcart Miller.
Mary Louise Neville.
Mary Pamela Short.
John Whitwell Hope Simpson.
Esther Mary Swinnerton.
Joseph Wilson Totten.
Arthur Harold Colyear Walker.

BOOK REVIEWS

"Techniques of Conception Control." By R. L. DICKINSON, M.D. The Williams and Wilkins Company, Baltimore, 1942. Price 50 cents.

THIS is a practical manual issued by the Planned Parenthood Federation of America, Inc.

A short foreword has been written by the Obstetrician-in-Chief of the Johns Hopkins Hospital, pointing out that the question of contraception is no new problem except in so far that it is now considered to be a medical one. He considers that all medical students should receive adequate training in methods of conception control so that they shall be able to deal with this question as it arises in the course of practice after qualifying.

This new and enlarged manual is the successor to "The technique of Contraception" by Eric M. Matsner, published by the National Medical Council of Birth Control.

The scheme of the manual covers the whole subject of contraception and also includes a short paragraph on the importance of the investigation and treatment of sterile mating.

The subject is approached from a scientific point of view intended for the use of those who have a good basis of anatomical and physiological knowledge as well as a certain degree of gynaecological experience. It is definitely not a book intended for the use of the lay public.

Chapters are devoted to the consideration of effectiveness and acceptability.

The various types of contraceptive appliances available for female application are described with a review of the advantages and disadvantages attached to their use. The place of chemical contraceptives and douches is considered as well as the place of the male condom, and the practice of withdrawal.

There are, in addition, chapters on the safe period, prolonged protection with a warning against the use of intra-uterine stems, sterilization without unsexing, and the indications for contraception.

The whole manual leaves very little to the imagination as details are given as to the materials and standards of manufacture of contraceptives with suggestions that there should be agreement as to standards of quality, co-operation among manufacturers and dealers to maintain quality, inspection and reports, lists of trustworthy makes and makers and dealers, regularly issued, with provision for wholesale or reduced rates to clinics and the poor.

Illustrations are 50 in number; of these 45 are most beautifully executed life-size drawings and the remainder are serial diagrams of the operations of vasectomy and salpingectomy.

The whole manual can without reservation be recommended for the instruction of the medical profession in this country as it appears to have been used in North America.

R. H. B. ADAMSON

"Textbook of Midwifery." By WILFRED SHAW. J. A. Churchill, London, 1943. Price 21*s*.

THIS is a book for students, written in the main on conventional lines. Illustrations are plentiful and good, many of them being taken from German sources.

There is internal evidence that the book has been put through the press in a hurry. Many redundancies should be cut out in a second edition. On the other hand there is only one brief allusion to the Rhesus factor. The relation between hydrops foetalis, icterus gravis, and congenital haemolytic anaemia is not mentioned.

Mr. Wilfred Shaw ends his introduction with the following admirable paragraph: "The best results in midwifery will surely be obtained by sifting cases at pre-natal clinics, by distributing normal cases to midwives, difficult cases to hospitals and the intermediate group either to general practitioners with special obstetrical qualifications or to obstetrical specialists. More than this is required, and adequate hospital accommodation is necessary. The maternity hospitals or the maternity wings of general hospitals should be of modern construction with opportunities for treating septic cases in isolation blocks, for barrier nursing and, what is particularly required at the present day, for nursing premature babies."

Review of Current Literature

Director: FREDERICK ROQUES, M.A., M.D., M.Chir. (Cantab.), F.R.C.S., F.R.C.O.G.

THIS Review contains the lists of contents and abstracts of the more important articles from the journals with which the *Journal of Obstetrics and Gynaecology of the British Empire* exchanges.

The Review of Current Literature has kept the readers of the Journal in touch with current literature throughout the world; owing to the war many journals with which the *Journal of Obstetrics and Gynaecology* previously exchanged are no longer received. At the end of the year an Index of all the subjects contained in the articles of the journals reviewed is printed. Arrangements are also made to include abstracts of important articles on borderline subjects, such as Physiology, Biology, and Biochemistry.

LIST OF ABSTRACTORS

J. LYLE CAMERON, F.R.C.S.
W. E. CROWTHER, M.B.
R. H. B. ADAMSON, M.D.
B. JEAFFRESON, F.R.C.S.
P. MALPAS, F.R.C.S.
T. N. A. JEFFCOATE, F.R.C.S.
MEAVE KENNY, F.R.C.S.
JANE H. FILSHILL.

The Lancet

January 16th, 1943.

*Failing lactation. Margaret Robinson.

A reflex governing the outflow of milk from the breast. H. K. Waller.

February 27th, 1943.

*Superficial atresia of the vulva. Constance M. Ottley.

April 24th, 1943.

*Effect of emotional shock on hormonal release and endometrial development. A. A. Loeser.

May 15th, 1943.

*Fatal air embolism after insufflation of the vagina. R. L. Brown.

July 17th, 1943.

*Effect of war conditions on the premature infant. C. Elaine Field.

FAILING LACTATION.

Margaret Robinson reports on a series of 1,100 consecutive cases of premature weaning. All cases of cessation of breast-feeding before the end of the seventh month were considered to be premature. About 40 per cent of the women could give no reason for the failure to continue breast-feeding and no reason could be found on examination.

The author describes four types of failure:—

(1) Dry, which may be partial or complete. The latter is the commonest type of all and is characterized by either a sudden or a gradual cessation of both the solid and liquid part of the secretion in one or both breasts.

(2) Wet. During the period of failure, or just before it, the mother complains that the milk has turned to "blue water." The breasts remain soft and there is a distressing lactorrhoea. The author thinks that this is due to overaction of the peristalsis of the ducts, preventing accumulation of the milk in the breast.

(3) Solid. In this type the breasts are not emptied and become knotty and hard. On expression only beads of thick creamy secretion are obtained. The absence of pumping makes it impossible to empty the breasts and manual expression nearly always results in abscess formation. The author is of the opinion that in these cases the milk solids are manufactured but the filling with fluid does not take place and so the pumping action cannot be maintained.

(4) Alactea. Complete absence of lactation.

When discussing the causes of failure, Margaret Robinson states that each lactation seems to be

peculiar to its previous pregnancy and that pregnancy and lactation should be considered together, instead of as separate entities. As each time a fertilized ovum is implanted in the uterus a fresh impulse is given to the system of ductless glands controlling lactation and this impulse persists until the baby can live separately from its mother.

She gives the reasons for weaning as: (*a*) the health of the mother, 23 per cent; (*b*) the condition of the infant, 12 per cent; (*c*) environment 8 per cent.

With regard to the health of the mother the author found that local conditions of the breast were more common than those affecting the general health of the mother. Most of 48 temporary failures were due to acute illness, the lactation cycle recurring with greater frequency and intensity as the severity of the disease abated. She found that menstruation was neither a reason for weaning nor a cause of failure; its function seemed to be independent of lactation.

With regard to environment, illegitimacy was the most important cause, and in 30 of these 38 cases weaning was due to the mother's having to work. Other reasons were illness at home and worry.

With regard to conditions affecting the baby, illness of the child and prematurity were the most frequent reason given for weaning. The author believes that the greater the prematurity the less milk is secreted per feed and the shorter the lactation period.

Superficial Atresia of the Vulva.

Constance Ottley reports two cases of this rare condition. The first case was that of an unmarried woman of 49 years, who had noticed a gradual closure of the vaginal orifice since the menopause two years previously; the second case was that of an unmarried woman of 91. Both of them had difficulty on micturition and both women developed acute retention of urine. On examination the cases were alike inasmuch as there was a complete absence of hair, the labia majora were absent, the clitoris was greatly hypertrophied, and the labia minora were fused in the midline except for a tiny opening just large enough to admit the point of a fine probe. In each case there was no difficulty in breaking down the line of fusion with the probe after it had been made to enter the minute orifice. The author points out that the adhesion of the labia minora appeared as though gummed rather than fused together. Neither patient had had any previous gynaecological condition. The author was quite unable to give an explanation for the hypertrophied clitoris which usually atrophies after the menopause.

Effect of Emotional Shock on Hormone Release and Endometrial Development.

Every healthy woman shows, at a certain time of her menstrual cycle, a certain endometrium. Loeser maintains that not only is the renewed birth, life and death of the endometrium subject to hormonal factors but to every kind of psychic factor also. He reports four cases in which amenorrhoea resulted from a definite psychic trauma—a state of affairs which had long been known to occur. He is of the opinion that the amenorrhoea is produced by a disturbance of hormone release.

In three of the four cases the emotional shock was due to the explosion of a bomb near-by and each occurred on a day within the first half of the intermenstrual phase. About 10 to 14 days after the missed period a biopsy of the endometrium was taken and on microscopical examination by Emil Novak the findings in each case corresponded to that day of the menstrual cycle on which the original shock occurred. The fourth patient was an unmarried woman, who feared she was pregnant following intercourse without her usual contraceptive. Biopsy of the endometrium after the missed period corresponded to the day of the menstrual cycle on which the physic trauma had occurred.

The author is of the opinion that the emotional shock must have had an effect *via* the autonomic nervous system on the release of the gonadotropic hormones and that this theory has been proved on histological examination.

Fatal Air Embolism After Insufflation of the Vagina.

One of the recognized methods of treating a trichomonas infection of the vagina is to insufflate with silver picrate powder and it is with consternation that one reads of a fatality due to air embolism. Everyone must feel grateful to Brown for reporting the case, especially as the insufflation was

performed during the later weeks of pregnancy. The booklet of indications and contra-indications supplied by the makers of the powder definitely states that the insufflation should not be done during menstruation or pregnancy. The opinion put forward by Brown that there must be an efficient cervical barrier present at the time of the insufflation is probably correct and this barrier would obviously be absent during pregnancy and menstruation.

EFFECT OF WAR CONDITIONS ON THE PREMATURE INFANT.

Elaine Field reports on a series of 185 cases of prematurity which included infants of 5½ pounds, or less, born in a London hospital after the declaration of war until August 1942. The stay in hospital was extended until satisfactory progress was made. All the infants were fed on breast milk and the total mortality was only 8.1 per cent.

This result compares most favourably with the high mortality figure of 50 per cent given in the literature during the last 12 months and on the figure of 20 per cent which, in the opinion of the British Paediatric Association, should be achieved by special paediatric care, supported by skilled nursing in suitably equipped units.

The author lays much stress on the nursing technique from the moment the infant is born. The body temperature was regulated by an electric blanket and as little handling as possible. The cord was only dressed every third day and the baby oiled twice a week. After the birth weight had been ascertained no further weighing was allowed until the fourth week. Feeding was begun 8 hours after delivery with a feed of breast milk given with a pipette and diluted with Hartmann's Ringer-lactate solution. By the seventh day whole breast milk was usually tolerated. The infant was put to the breast as soon as the sucking reflex was established, usually when about 5 pounds in weight. She does not agree with oesophageal feeding because of the risk of aspiration pneumonia and because it does not stimulate sucking. In addition to oxygen, dry extract of thyroid, gr. 1/10, was given twice a day for the first two weeks, and in the second week iron was commenced. By the end of the month vitamins A, D and C were included.

The author maintains that the second week is the critical period for these premature infants, as they become reluctant to feed and therefore dehydrated. She recommends at this stage one or two subcutaneous injections of 50 c.cm. of 5 per cent glucose in Hartmann's solution and states that it may save many lives. BRYAN JEAFFRESON

The Canadian Medical Association Journal

July 1943, Vol. xlix, No. 1.

*Obituary notice.

OBITUARY NOTICE.

Dr. Allan Roy Dafoe died on 2nd June, 1943, at North Bay, Ontario, Canada, at the age of 59 years. The cause of death was pneumonia.

Dr. Dafoe was known throughout the world as the " Quins' Doctor," having delivered Mrs. Oliva Dionne of Callandar, Ontario, of quintuplets on May 28th, 1934. It seems entirely due to his assiduous care that these tiny and premature baby girls survived.

This famous doctor was born in the village of Madoc, near Belville, Quebec, on 29th May, 1883. He was the son of a country doctor. Graduating from the Toronto University in 1907, he went to the lumber town of Callandar, Ontario, where he practised for 27 years in the obscurity of the country.

He was Medical Officer, under the guardianship of the Ontarian Government, of the quintuplets from their birth until 1942, when he resigned the post. To the end he gave them the same affectionate and unremitting care that he administered in their earliest hours, when their little lives hung so precariously by so frail a thread.

J. LYLE CAMERON

Amatus Lusitanus, Lisbon

May 1942, Vol. i, No. 6.

*Pregnancy and valvular heart lesions. Caldas.

Estimation of food supply available for the population of Portugal. Loureiro.

*Infiltration of the pelvic-perineal plexus in spastic conditions of the uterus in labour. Almeida.

Clinical notes. Bifid urethra in a woman. Macedo.

PREGNANCY AND VALVULAR HEART LESIONS.

Caldas discusses the prognosis and treatment of cardiac lesions during pregnancy. In pregnancy a woman suffering from cardiac valvular lesion is subject especially in parturition and, to a lesser degree in the puerperium, to circulatory disturbances often intense, while at the same time she is threatened with purely obstetric complications.

When the heart has sufficient energy to undertake the greater work requisite, the prognosis will be good, but will be grave when the heart energy is or grows insufficient for added work. "According to the state of the myocardium so will evolve the pregnancy" (Porak).

What is of value is not the anatomy of the myocardium but its functional power. Sir. J. Mackenzie thinks functional criterion is the best test of heart efficiency, the anatomical may induce errors in prognosis and should be abandoned. The woman's normal activity represents the best functional test. In accordance with this basis, the American Heart Association has established a classification of cardiac conditions in pregnancy, almost generally accepted:

(1) No limitations of physical activity. Usual exertions cause no disturbance. Objective signs form the only evidence of heart disease.

(2) Slight or moderate limitation of physical activity. Habitual exertions of strength are not well borne.

(3) Great limitation of physical activity. Exertions even less than usual are ill-supported.

(4) Incapacity for any physical activity without feeling ill. There is complete loss of compensation.

Caldas publishes tables of mortality in the various conditions, which prove the reliability of the American classification. He gives therapeutic measures employed by himself and others, including prenatal care, and the treatment of mild cases, which become serious, as well as of cases which are grave from the beginning. When interruption of pregnancy must be carried out, he recommends operation *per vaginam* in the early months. In the later months abdominal operation produces less circulatory disturbance.

INFILTRATION OF THE PELVIC-PERINEAL PLEXUS FOR SPASTIC CONDITIONS OF THE UTERUS IN LABOUR.

In 1938 Henriet published his results in the treatment of uterine spasms in labour, by anaesthetic infiltration of the pelvic sympathetic nerves. He thought that, by this process, relaxation of the cervical muscular fibres would be obtained by a preliminary paralysis of branches of the sympathetic nerves related to the cervix. The extreme simplicity of technique, harmlessness of the method and, above all, its successful results induced Almeida to adopt the same practice. To-day, after 4 years of its employment in many private and hospital cases, he considers his results equally successful. In very few cases were there negative results. In most, the results were excellent in cases which might have required incision of the cervix, forced dilatation or other methods equally disagreeable.

Antispasmodics used early may relieve some cases, but when these fail, resort to infiltration is valuable. Even when dilatation is not complete after 10 minutes of infiltration, the cervix or the contraction ring, passes into such a degree of relaxation that extraction of the foetus can be easily carried out.

Almeida briefly describes composition, from many origins, of the uterine nerve fibres, proving that they branch to all regions whence cervical complications may arise.

He describes the technique employed in lateral injections of novocaine practically the same as that of Henriet and tabulates the results obtained in four cases.

These observations show that dilatation usually occurs rapidly. He calls attention to phenomena described in the fourth case. On making the left infiltration after the right, and then placing his hand near the contraction ring, he detected relaxation in the right half before that in the left, showing that innervation of one side is the independent of the other.

Summing up his own results, Almeida concludes that:

(1) Infiltration of Frankenhäuser's plexus is innocuous and the technique is simple.

(2) In more than half of the cases of spastic conditions of the cervix or of contraction ring there was rapid dilatation, easily permitting the application of the forceps, or internal version.

(3) A negative result of the method occurred in a very small number of cases.

J. H. FILSHILL

Medicina Española, Valencia

April 1943, Vol. ix, No. 51.

*Climacteric psychosis. Alcober.
*Caesarean section and transverse lie. Pastor.
Remote results in Curie-therapy of uterine cancer. Torro.

Climacteric Psychosis.

From the undisputed fact that the majority of women suffer from physical disturbances during the menopause, it is claimed that the psychosis which appears during this period is due to the cessation of follicular ovarian activity.

"In no problem," says Kehrer, "is structural analysis more justified than in the psychosis of the last third of life."

Quoting Sabatini, who published a monograph in 1938 on the pathology of the menopause, Alcober points out that a better individualization of causes is requisite. Psychosis arising during this period cannot be regarded as solely due to cessation of ovarian activity, but to this associated with senile or pre-senile changes present in both sexes. In both, corporal habits are modified, and vascular elasticity, so important in relation to age, is more rapidly lost in men than in women. Senile and pre-senile involution causes arterio-sclerosis and affects all endocrine organs. Alcober instances the frequency of similar psychosis, e.g., neurasthenic depression in men between 40 and 60. In such cases there exists a predisposition, not previously recognized, of physical and psychic character. When joint causal factors of sex and age are taken into account, it is not a question of menopausal but involution psychosis.

The surgical removal of ovaries gives rise to a most acute menopause, but not always to mental disorder. When this does occur there is usually a history of previous nervous instability, and this may not show itself till some time after operation.

Alcober records a case of a patient whose ovaries were removed in 1934. No mental disturbance appeared till 1939, and the symptoms did not differ from current schizophrenia.

He concludes that the existence of psychosis essentially dependent on the menopause cannot be admitted but that it may be a releasing factor of disorder in other endocrine glands than those of the ovaries, and to degeneration due to age.

Caesarean Section for Transverse Lie.

In a previous article Pastor advocated external version in cases of transverse lie, when the condition was recognized in the early months of pregnancy. Even then version might be difficult in certain circumstances.

He now discusses and compares proceedings to be adopted during the later months and in labour, internal version and forceps extraction or Caesarean section.

Though no extremist as regards employment of Caesarean section in all cases of shoulder presentation, he thinks that each case needs careful and thorough consideration before attempting version and vaginal extraction. Age, parity, elasticity of genital tissues, contraction of uterus, escape of amniotic fluid, narrowness of pelvis and sepsis are all factors which may prove obstacles and have dangerous consequences; such as perineal tearing extending to the uterus and leading to its rupture.

On the other hand, reviewing the statistics of many surgeons regarding low transperitoneal Caesarean section, he finds the mortality is nearly, if not quite, as low as in Caesarean section for other causes.

He gives Winter's statistics 2.2 per cent; Schroeder's and Holland's 1.8 per cent; Ken Stöckel, and Walthard, 0 per cent; and quotes Hirsch, "In future every case of transverse lie will be treated and terminated by Caesarean section."

J. H. Filshill.

REPORTS OF SOCIETIES

PROCEEDINGS OF THE ROYAL SOCIETY OF MEDICINE

(SECTION OF OBSTETRICS AND GYNAECOLOGY), MARCH 1943.

PROGRESS IN OBSTETRICS AND GYNAECOLOGY DURING THE PRESENT CENTURY.

(Presidential address by J. M. MONRO KERR, LL.D., M.D., F.R.C.O.G.)

Dr. J. Munro Kerr's presidential address to the Section of Obstetrics and Gynaecology of the Royal Society of Medicine is notable, as might be expected, for its wisdom and not devoid of pungency in expression. It should be read in full by those who can, but for others we abstract some of the arguments and conclusions which the author sets forth. Kerr surveys the past 40 years' advances, discusses present problems and sets forth his own suggestions for advances in the future.

Of the two great discoveries of this century, hormones and vitamins, it is concluded that practical application of the former to obstetrics and gynaecology have been in the main disappointing. Dr. T. Moore, Director of the Dunn Nutritional Laboratory at Cambridge, has written in response to an enquiry that it is inadvisable to give during pregnancy, particularly if the diet is low in vitamin B and E, massive doses of cod-liver oil, which in animal experiment appears to reinforce the effect of vitamin E deficiency in causing muscular dystrophy of premature labour. He also says that excess of vitamins during pregnancy will not cause supernormal growth, although a lack may reduce growth unnaturally: dietetic attempts to limit the foetal dimensions in cases of pelvic abnormality should include in reduced food quantities an adequate vitamin content. Kerr foresees a great future for block anaesthesia in surgery. While admitting the revolutionary changes brought by radium and deep X-rays in treatment of cancer of the cervix, he cannot convince himself that Wertheim's operation should be discarded. Nor can he discover that the problem of the aetiology of eclampsia is much nearer solution than it was 50 years ago. He stresses the beneficial effects of the founding of the Royal College of Obstetrics and Gynaecology and of the various Midwives's Acts.

Coming to present problems, Kerr contrasts the diminution of maternal mortality from sepsis with the insignificant decrease since 1925 in the toll of deaths from the toxaemias and haemorrhages and from the complications and accidents of labour: the reason is still to be found, he thinks, in the inadequacy of antenatal care and the too infrequent treatment of the abnormal pregnancy and complicated labour in special institutions by specialist physicians. Many foetal deaths, too, are due to faulty technique on the part of a doctor inexperienced in obstetric practice; and if one takes a broad view of gynaecological disability the aphorism is more true than most that "good obstetrics is preventive gynaecology." In a brief allusion to forensic aspects of abortion Kerr states his belief that it is undesirable to press for notification, and that the law should be left as it is. "Few if any of us are prepared to recommend extending the scope of the operation on economic or compassionate grounds as represented by the patient.

There are still fresh in our minds the disastrous results following the free employment of induction of abortion in U.S.S.R.—results so disastrous that U.S.S.R. gave up the experiment." There is little prospect of bringing about the urgently necessary increase in the birth-rate without State action directed to removing social obstacles to free expression of the parental instincts: Kerr has hard words to say of the vision and foresight of politicians in this connexion. An improvement in neonatal mortality and stillbirth mortality could be accomplished, however, by a national maternity service "in which obstetricians and the Public Health Service (with all its ancillary agencies) would be in full and sympathetic co-partnership. The sands are running out—no longer may we procrastinate and

set up committees of enquiry—impending disaster is on our doorstep!" Surveying the future Kerr forsees immediate or almost immediate efforts to organize a national maternity service—a service conducted by obstetric specialists and highly-trained midwives in well-appointed maternity institutions. He favours expert and institutional treatment for the wives of the artisan and the black-coated worker, as well as for the well-to-do, and those near the poverty-line. The work would be taken away to a very great extent from the general practitioner, whose compensation, it is maintained, would be found in a higher usefulness in pure medicine, and more especially its preventive aspects: details of financial compensation, however, are not suggested. The cities would have antenatal clinics and maternity hospitals or units of general hospitals, all staffed by specialists: in small towns and in the country local practitioners would elect certain of their number to specialize in similar work after taking postgraduate training and a diploma. Kerr advocates two Central Directing Bodies to direct regional administrative bodies in England and Wales and in Scotland—*ad hoc* central bodies of which the chairman should be the permanent secretary or chief medical officer of the Department and the membership should include representatives of regional and local authorities as well as nominees of the R.C.O.G. and B.M.A., midwives and insurance commissioners.

W. E. CROWTHER.

ROYAL ACADEMY OF MEDICINE IN IRELAND.

A Meeting of Section of Obstetrics was held in the Royal College of Physicians on Friday, October 15th, 1943. Dr. A. W. Spain was in the Chair.

Dr. NINIAN FALKINER reported

A CASE OF PLACENTA PRAEVIA TREATED BY CAESAREAN HYSTERECTOMY.

The patient, aged 42, was admitted to the Rotunda Hospital on September 14th as a case of antepartum haemorrhage. She was a multipara in her sixth pregnancy, having had 4 full-time deliveries and one abortion. Her general condition could only be described as desperate. She was in an advanced state of emaciation which subsequently proved to have been due to malnutrition. Her weight was 82 lb. Her previous history revealed that she had had infantile paralysis in childhood which had rendered her left leg atrophic. She was seen by both Mr. Pringle and Dr. Dockeray with a view to excluding a wasting disease but there was no surgical or medical condition found to account for her wasted condition. Her blood-pressure was 90/65 mm. hg. Her blood-count was 3 million red cells and her haemoglobin 57 per cent. Obstetrical examination showed that the case was one of lateral placenta praevia; the patient had been in the hospital for 8 days before a recurrence of bleeding took place. At this time the cervix was closed, the vertex was presenting, and the lower pole of the placenta could be felt on vaginal examination. It was apparent that any obstetric procedure to deal with either antepartum or postpartum haemorrhage would be fraught with grave risk and it was determined that her best interests would be served by doing a Caesarean hysterectomy, for by so doing both antepartum and postpartum haemorrhage could be reduced to an absolute minimum.

Operation. The anaesthetic employed was gas and oxygen. A mid-line incision was made, large enough to allow the uterus to be delivered. An incision was then made in the fundus of the uterus running transversely from one cornu to the other. The baby was extracted in good condition and then total hysterectomy was carried out. The abdomen was then closed and the patient made an uneventful recovery and left hospital on the 13th October. Her weight was then 81 lb. Her baby has done well; it weighed 6 lb. The blood loss during operation could not have exceeded 6 ounces, and all came from the placental site.

The specimen. The uterus was fixed in formalin; it was opened some days later and the placenta had not become detached. An interesting picture

of the detached lower pole of the placenta was demonstrated. The appearance suggested that the blood the patient was losing prior to and during the operation was blood that had passed through the placenta and was not coming directly from the uterine veins or arteries.

The PRESIDENT discussed the pressure methods of treatment. The amount of pressure necessary is very small to check the bleeding in placenta praevia. As regards general treatment there was a happy result in this case but he thought, on the whole, it would seem perhaps more radical than he would like to attempt himself. He asked was there any evidence that removal of the uterus was really necessary in this case. He would have recommended the lower segment Caesarean section in this case.

Dr. J. S. QUIN discussed the question of the amount of haemorrhage that occurs in Caesarean sections. He did not understand why a fundal incision should have been chosen unless Dr. Falkiner was afraid of making an incision over the placental site. Would he justify the fundal incision as against the classical incision or the lower segment incision? He considered that postpartum haemorrhage is the important loss. He said he had done a hysterectomy without opening the uterus and there was practically no bleeding.

Dr. J. F. CUNNINGHAM said, with regard to the origin of the haemorrhage, it comes from the placenta itself in these cases. In certain cases it must also come from some of the sinuses that are exposed. Haemorrhage comes from both places. In lower segment Caesarean sections the amount of haemorrhage depends on a number of factors. He said he had done a classical section in the presence of prevesical veins in the anterior part of the lower segment, as haemorrhage from there is difficult to control. He did not think the treatment in this case should be generally followed in cases of placenta praevia but he was sure it was correct in Dr. Falkiner's case on account of the patient's general condition of emaciation, and that he was justified in his judgment.

Dr. BETHEL SOLOMONS said that, although there was no question of Caesarean hysterectomy being recommended as a routine for placenta praevia, in this case it appeared to be correct. He thought that Dr. Falkiner should bring further proof of his contention that all the haemorrhage in placenta praevia came through the placenta; the half-section of the uterus presented did not show this. Pressure of the infant on the placental site was very necessary after bipolar version, as proved by the fact that if pressure was relaxed the haemorrhage recurred. He suggested that in the case reported, haemorrhage might have been further avoided by removing the foetus and uterus without opening the latter; he had seen Potter extract a live foetus after Caesarean hysterectomy.

Dr. R. M. CORBET said he had seen this patient with Dr. Falkiner after the operation and there was no doubt that any small haemorrhage would have been fatal. The procedure, therefore, was correct, with the exception that he took a considerable risk in examining the patient vaginally. He agreed with lower segment Caesarean section in cases of placenta praevia. He said he used to do a classical Caesarean section but was later converted because bleeding, which one could not see, went on after the classical incision.

Dr. FALKINER replied. He said the criticisms were quite justified. He was faced with a woman who, he thought, would be in a very dangerous position if she lost blood. He treated her by this method and there was very little blood in the fundal incision. With regard to the source of the blood, there was no doubt that the bleeding which was taking place when she came to the theatre, was blood which had passed through the placenta.

Doctors EDWARD SOLOMONS and NINIAN FALKINER reported

A CASE OF CALCIFICATION OF THE PLACENTA

The placenta was exhibited and microscopic sections showed the details of the condition. An X-ray photograph also showed the extensive calcification.

The patient was a primigravida, aged 22. The pregnancy was normal. Labour commenced during the forty-first week and was quite uneventful until the membranes protruded at the vulva at the end of 24 hours; when artificially ruptured solid meconium came away. As the head was on the perineum it was quickly and easily delivered with forceps. The foetus was stillborn. Free bleeding occurred after delivery and it was necessary to express the placenta 15 minutes later. The puerperium was normal.

Examination of the placenta revealed small, hard masses, each about the size of a pin's head spread over the entire maternal surface of the placenta.

Dr. FALKINER demonstrated the placenta in Dr. Solomons's case. The placenta showed extensive calcification which was widely spread through the villous tree. There was no suggestion of any calcification in the maternal part of the placenta. Microscopic nodules of calcium apparently scattered over the maternal surface of the placenta were definitely shown to be in the walls of foetal vessels. This position of the calcium helps to demonstrate the truth of Spanner's contention that large foetal vessels traverse the intervillous spaces passing right down to the basal plate before splitting up into their terminal branches.

The PRESIDENT said this case opened up possibilities when death of the foetus occurs without reason at the end of labour. Perhaps the pathologists will throw light on the reason for the deposit of calcium in the placenta. How can it be prevented? And how frequently does it occur?

Dr. J. S. QUINN asked if the child's parathyroid glands were examined postmortem and was calcification found in other blood vessels of the child. The mother's blood calcium was said to be 10.8 mgm. per 100 c.c.: was that long after delivery?

Dr. F. W. DOYLE described a case of his in which similar calcification had been found. It was a case of breech presentation with extended legs. Delivery was carried out under gas and oxygen. There was not any trouble or haemorrhage.

Dr. A. W. SPAIN and Dr. GEOGHEGAN described

A CASE OF CHORIONEPITHELIOMA

in a woman, aged 30, who had been delivered of a living child at term on February 7th, 1943. She had menstruated regularly in March, April and May, but had bled *per vaginam* from May 18th to July 14th, after which she had continued to bleed until August 7th, when she was readmitted to hospital. After this she had been bleeding and a Zondek-Aschheim test was found to be positive. Panhysterectomy was done on September 17th. On October 4th she was discharged from hospital apparently well.

Dr. Spain gave the history of the case and Dr. Geoghegan described the pathological findings and showed the slides.

Dr. R. M. CORBET asked why Dr. Spain sent the urine to Dr. Dockeray for the Zondek-Aschheim test, instead of doing the Hogben test himself. He asked Dr. Spain when the patient last ovulated. Should a patient who has chorionepithelioma sufficiently marked to give positive reaction be able to ovulate?

Dr. J. F. CUNNINGHAM said he felt the diagnosis of chorionepithelioma was not an easy matter. In 5 cases of these which he had seen, 3 followed operation. He described a case of his which he had treated by deep X-ray. Later a secondary growth occurred in the anterior vaginal wall. He also described another case of a dead foetus with chorionepithelioma in the uterus. He thought that the Zondek-Aschheim test was useful in diagnosis and in treatment. He did not think X-ray therapy was really necessary, because most of these tumours that are removed are not likely to continue to grow.

Dr. G. C. DOCKERAY said they had had 6 or 8 of these cases in the Rotunda Hospital. It may be possible to make the diagnosis histologically, but if the growth is deep in the muscle it is possible to miss chorionepithelioma. It is considered that malignant villi are present. There are no villi in chorionepithelioma.

Dr. N. McI. FALKINER pointed out that these tumour cases *do* occur after full-time pregnancy He discussed the pathology. He said he wondered if some of them originate in early pregnancies or in missed abortions. It is shown definitely that these cases follow full-time pregnancy.

Dr. SPAIN replied. He said he did not depend on the Hogben test, because he considered Zondek-Aschheim reaction more reliable. He did not know when the patient ovulated last; it seemed that she did so before the March, April and May periods. The tumour seemed to become active after the period in May. In the early stages it inhibited menstruation. He was grateful to Dr. Cunningham for his opinion about the question of deep X-ray therapy. It was, in his opinion, difficult to decide whether this condition more frequently follows abortion, full-time delivery or hydaordiform mole. According to Novak 50 per cent occur after moles and 50 per cent after pregnancies. Clinical signs and symptoms are of great importance in these cases.

Dr. BETHEL SOLOMONS reported:

A CASE OF INTERSTITIAL TUBAL PREGNANCY.

He said that Mrs. S., aged 24, menstruated from June 1st to June 6th, she married on June 9th, and menstruated from June 28th to July 3rd; then very slightly on August 4th and 5th: eleven days later she had a heavy bleeding, with clots, after some horse exercise. On August 24th Dr. Delia Horne saw her and found that the uterus and cervix were soft; she was suspicious of pregnancy, but the Zondek-Aschheim reaction was negative. Curettage was done on September 1st. As the patient was still complaining, Dr. Horne asked Dr. Solomons to see her in consultation on September 28th; when the uterus was found to be retroverted with what felt like a painful right ovary, but being suspicious of ectopic gestation, laparotomy was recommended. At the laparotomy the caecum was found adherent to a mass in the right uterine cornu. The intestine was easily separated. The right Fallopian tube with an interstitial pregnancy was removed; which necessitated resection of part of the right cornu of the uterus. The raw surface was covered and the abdomen was closed. The ovary was left *in situ*. An interesting point to refer to here was the negative Zondek-Aschheim reaction. Evidently a negative result does not contra-indicate ectopic gestation. The condition is extremely rare. Teacher states that, "Interstitial pregnancy includes only 3 per cent of all cases of ectopic pregnancy." As ectopic gestation occurs about once in 400 births in hospital, the general opinion is, that unless the ovum dies, intra-abdominal rupture will occur with fatal results. Abortion into the uterus has never been proved to have occurred, and a definite diagnosis of interstitial pregnancy is difficult to make, although it obviously would be suggested by the presence of a mass close to the uterine cornu, together with a history of ectopic gestation. The outlook for a future intrauterine pregnancy is good.

The PRESIDENT was interested in this rare condition. He said there was great difficulty in diagnosing this from an angular pregnancy.

Dr. G. C. DOCKERAY discussed the pathological aspect of the case and the fact that the Zondek-Aschheim reaction was negative. He considered that the ovum must have been dead for some time on account of the large inflammatory cells. Many were plasma cells which showed that the ovum must have been dead, thus accounting for the negative Zondek-Aschheim test.

Dr. N. McI. FALKINER described a case he had brought to the Academy last session when apparently an interstitial pregnancy had continued to term. The placenta was adherent and had to be separated manually at laparotomy and a full-time live child was removed.

Dr. R. M. CORBET described a case of interstitial pregnancy he had seen some years ago. The whole cornu of the uterus was the site of an abscess. He did a hysterectomy.

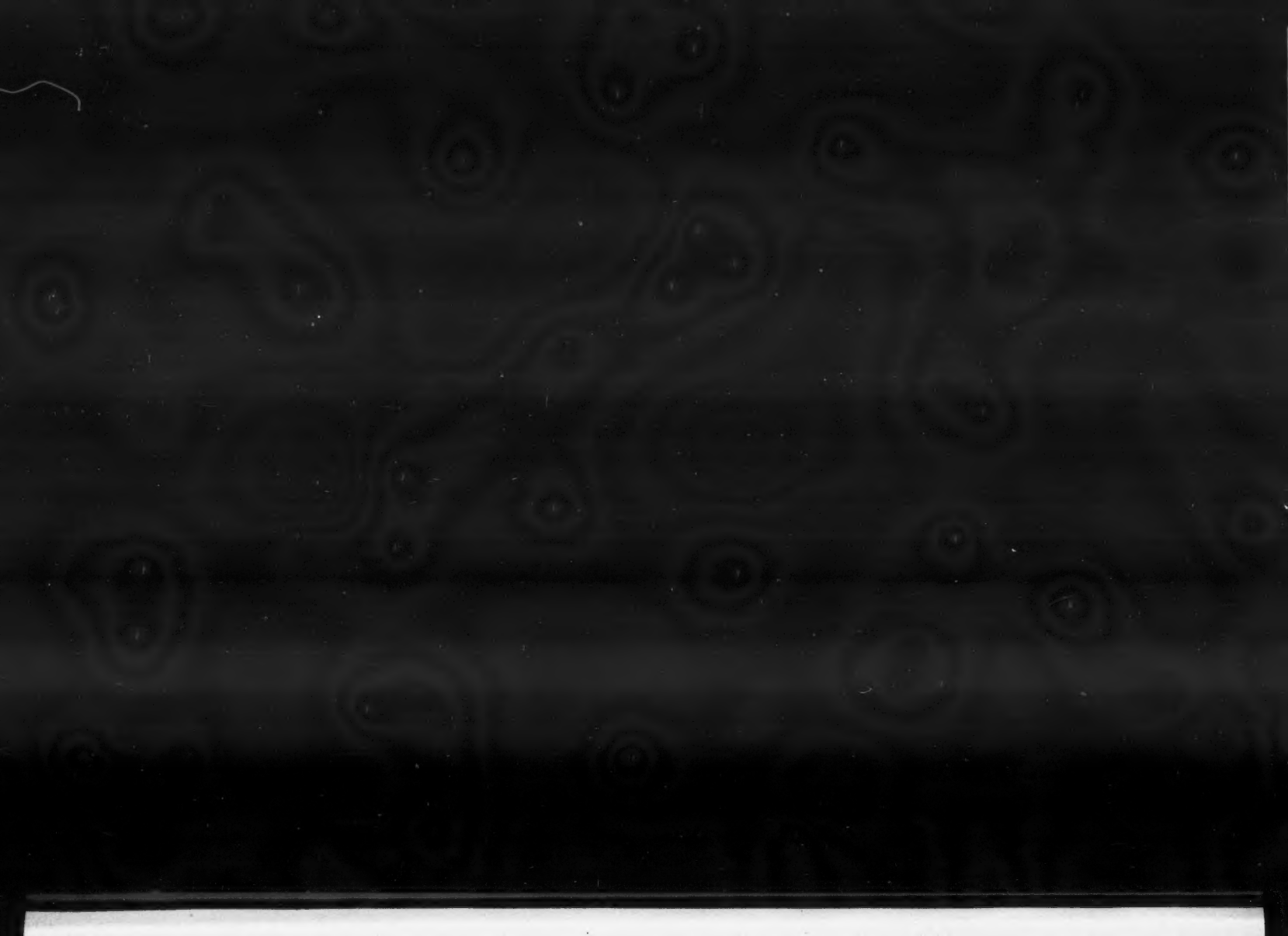
Dr. SOLOMONS replied. He agreed with Dr. Spain about the difficulty of diagnosing a deflected ordinary pregnant uterus from a tubal pregnancy. He was interested to hear there was other cases of full-time ectopic pregnancies in the National Maternity Hospital. He considered the patient ought to have a normal confinement on the next occasion.

Dr. R. M. CORBET reported:

A. CASE OF CARCINOMA OF THE FALLOPIAN TUBE

ERRATUM.

In the Report of the Royal Academy of Medicine in Ireland, published in the August issue of the Journal, page 315, Dr. Corbet is quoted as saying that the incidence rate of eclampsia in the Coombe Hospital was 5.3 per cent. The figure should have read 0.53 per cent, and the publishers regret the error.



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